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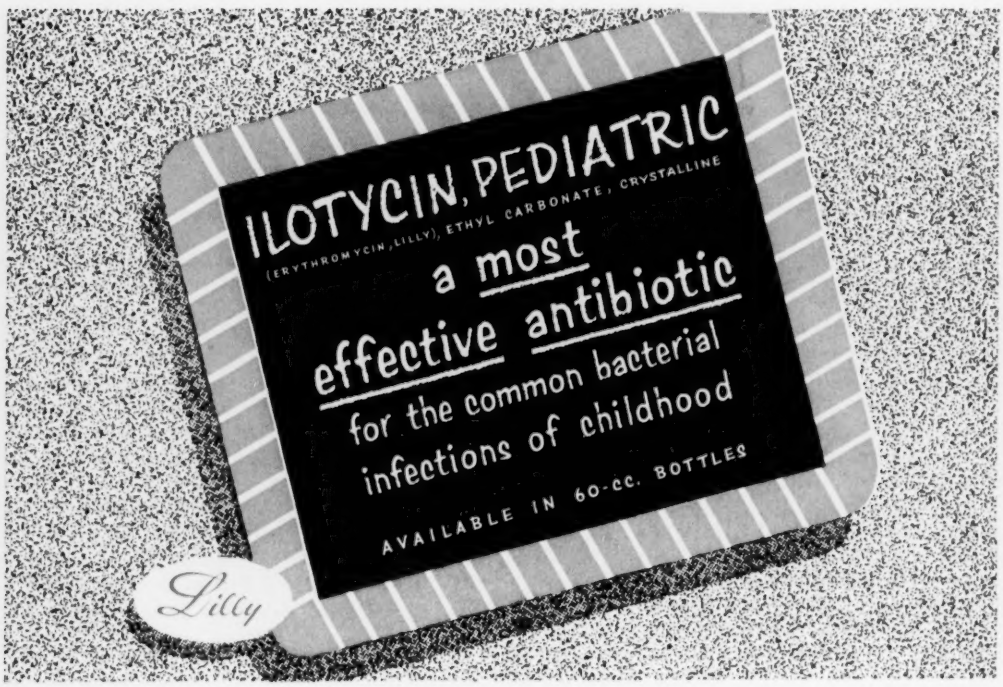

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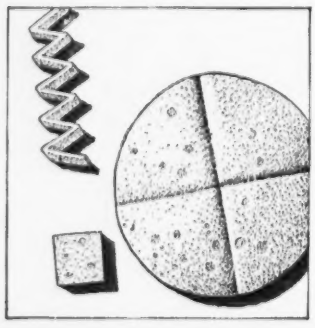

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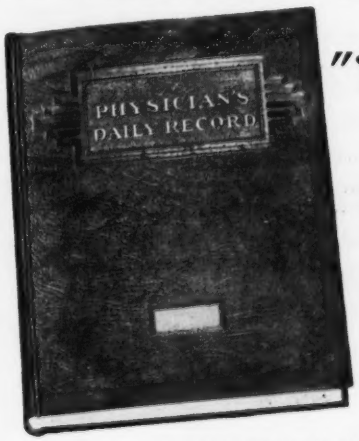

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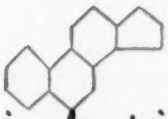
The symbol SCHERING denotes leadership in steroid hormone  research and manufacture -- including Cortogen in the most useful clinical forms. For systemic therapy, use Cortogen tablets SCHERING or injection . For eye disorders use Cortogen ophthalmic suspension -- sterile . The Schering seal SCHERING appears on each tablet and package -- your assurance of quality.

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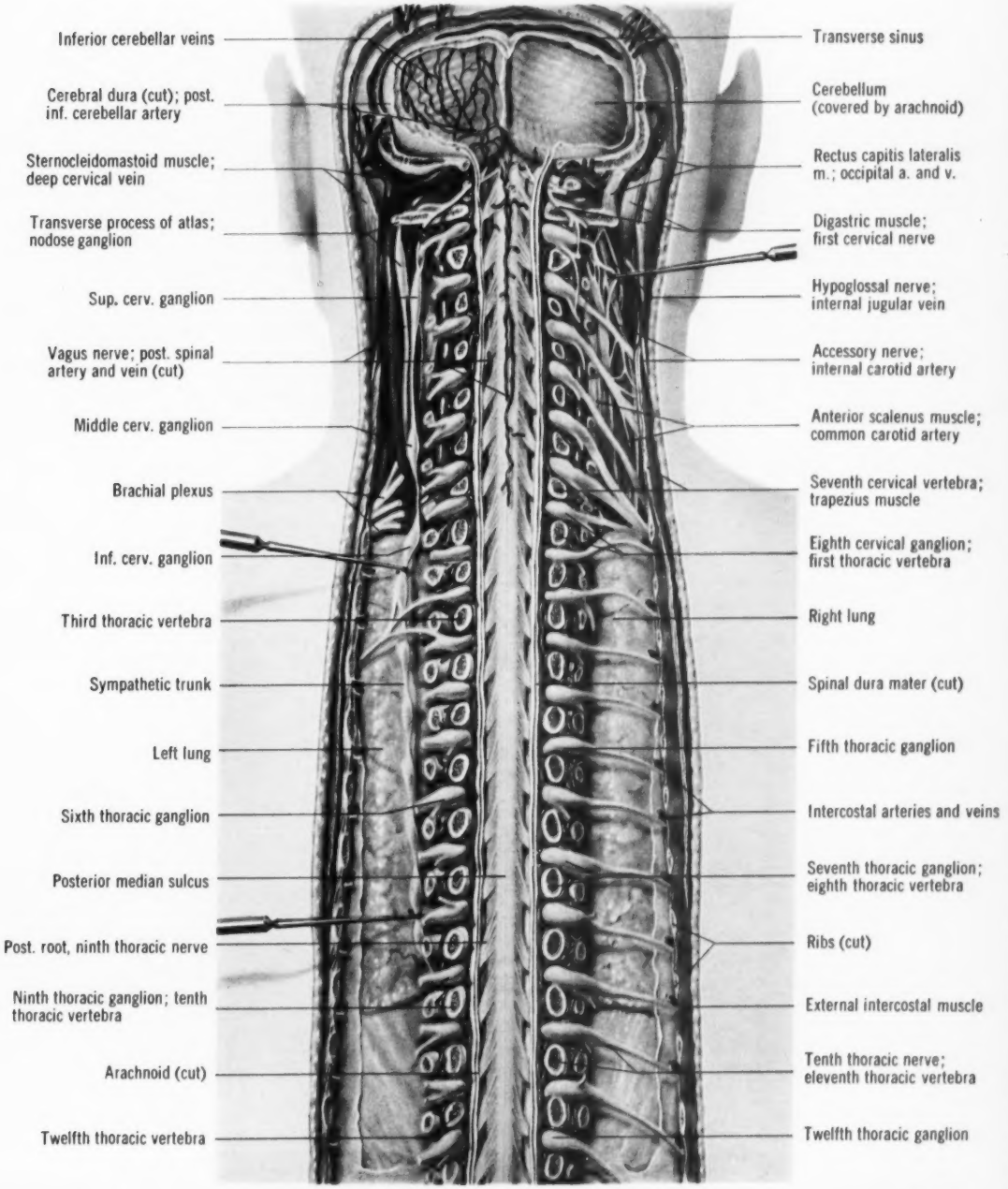
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Third thoracic vertebra
Sympathetic trunk
Left lung
Sixth thoracic ganglion
Posterior median sulcus
Post. root, ninth thoracic nerve
Ninth thoracic ganglion; tenth thoracic vertebra
Arachnoid (cut)
Twelfth thoracic vertebra
Transverse sinus
Cerebellum (covered by arachnoid)
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Digastric muscle; first cervical nerve
Hypoglossal nerve; internal jugular vein
Accessory nerve; internal carotid artery
Anterior scalenus muscle; common carotid artery
Seventh cervical vertebra; trapezius muscle
Eighth cervical ganglion; first thoracic vertebra
Right lung
Spinal dura mater (cut)
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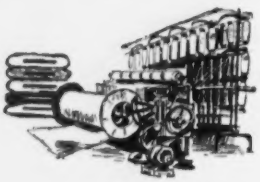

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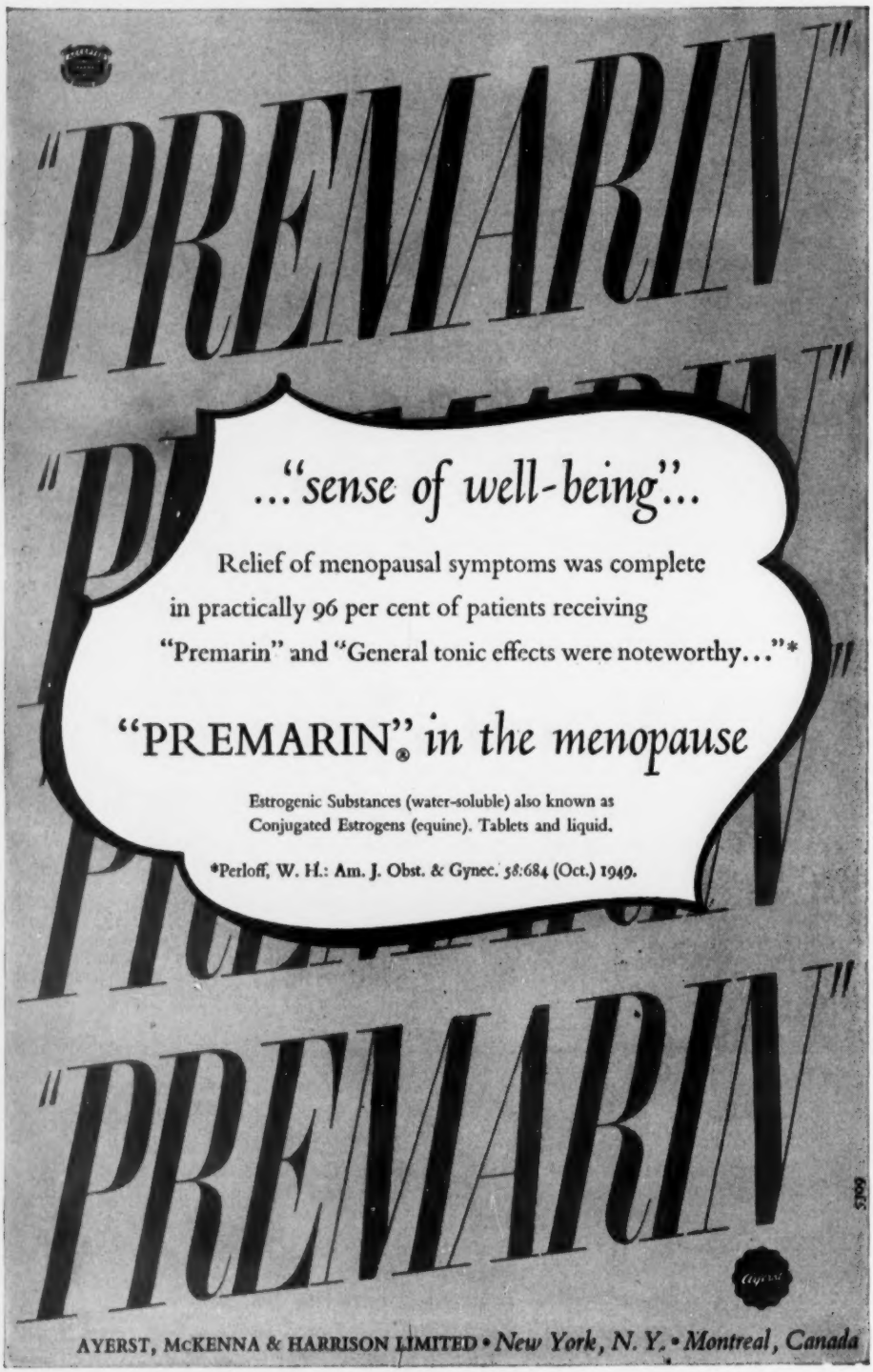
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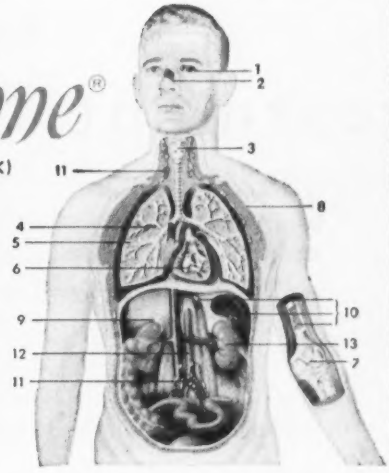

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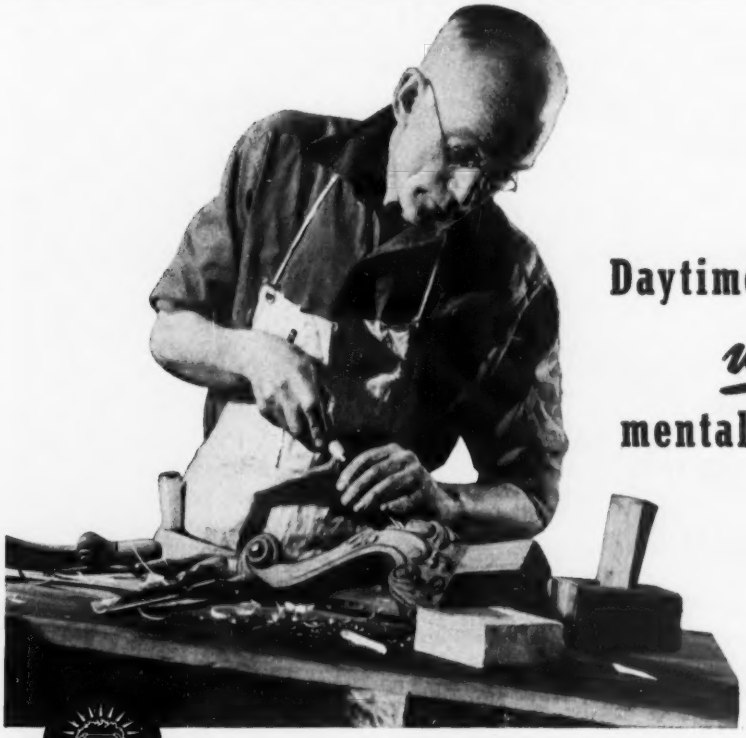

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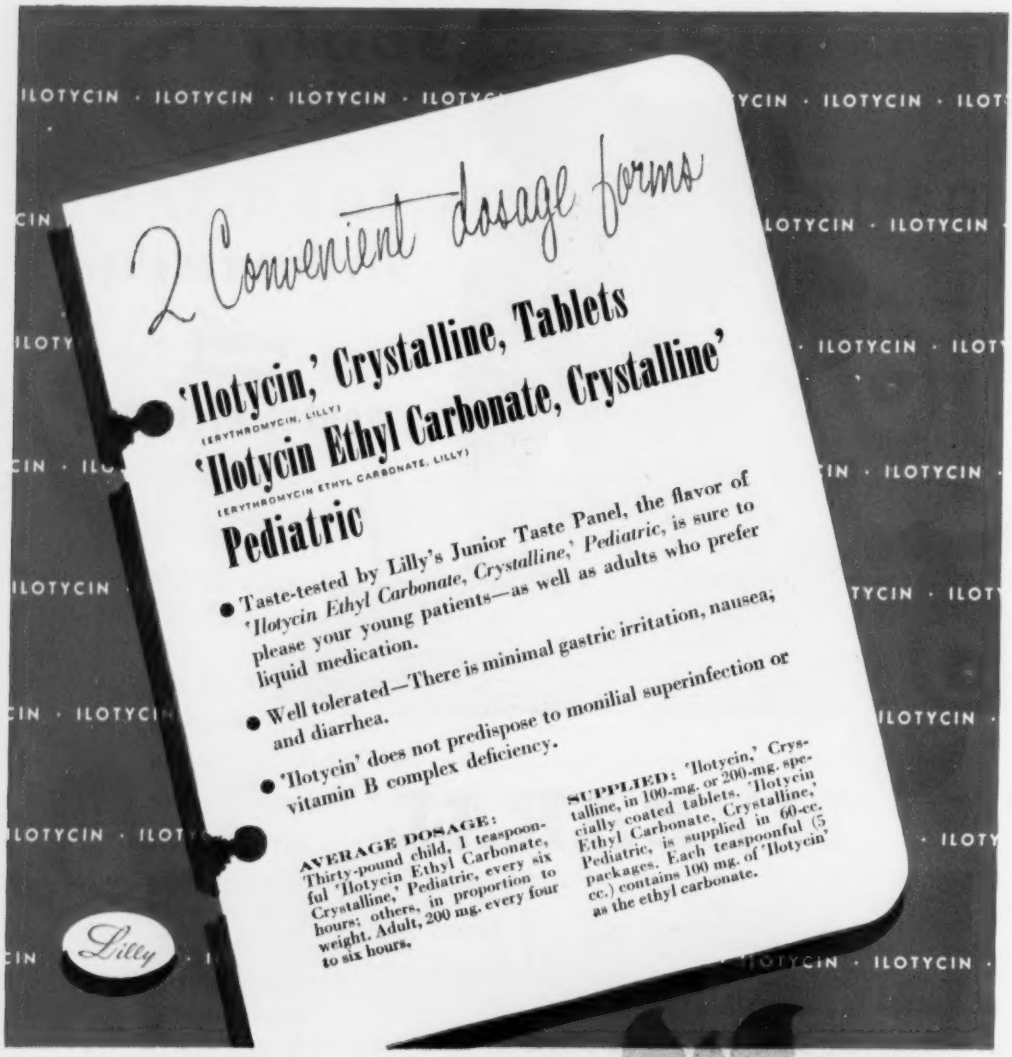
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VOLUME 14 OCTOBER, 1953 NUMBER 10

THE CLINICAL AND PHYSIOLOGIC ASPECTS OF EMPHYSEMA

Diagnosis and Treatment

BURGESS GORDON, M.D.

PHILADELPHIA, PENNSYLVANIA

The significance of emphysema is brought home with the increasing longevity of our population; and, as the special drugs gain new victories over pneumonia and the severe infections, the frequency of this very disabling and challenging disease will probably be even higher. The presence of emphysema with cardiovascular conditions in later life emphasizes the fact that tissue degeneration is an important contributing factor.

The aspects of emphysema are structural and physiologic. The complicating factor sometimes succeeds the basic processes and perpetuates the important disabling features. This is illustrated in cases of simple emphysema that become accentuated by superimposed bronchial disease.

Etiology

Emphysema is associated with impairment of the elastic recoil of the alveoli during expiration. Contributing factors are congenital weakness or defect of the elastic tissue of the lungs; degenerative changes associated with chronic bronchial and pulmonary infections; bronchospasm in asthma; whooping cough; inhalation of dusts and gases that favor irritation of the bronchial mucosa and alveoli; extremely cold weather; silicosis; hard bouts of coughing; improper exhalation; pulmonary arteriosclerosis; failure of the heart; heavy lifting, and abnormal curvature of the dorsal spine[1]. Experimentally, moderate continued distention of the lungs will cause permanent loss of elasticity and disturbances of the circulation.

Read before the Second General Session, Medical Society of the State of North Carolina, Pinehurst, North Carolina, May 18, 1953.

From the Department of Medicine, Woman's Medical College of Pennsylvania, Philadelphia, Pennsylvania.

Pathologic Features

The production of emphysema is both a physical and a physiologic process. Structurally, the alveoli are stretched, thinned, and ruptured. Large irregular spaces are not uncommon in advanced cases. The capillaries running into the walls of the distended air cells are contracted or obliterated. The respiratory bronchioles are dilated, and not infrequently there is so-called focal emphysema.

The pathology of emphysema has been demonstrated interestingly by Gough[2] in paper-mounted large sections of lung. His preparations are representative of the whole lung, with indications of the over-all severity of the emphysema. Since the condition is rarely uniform throughout, the study of selected areas alone may give a misleading impression of the total lung damage.

The patterns suggest that the mechanics of production are of at least two fundamentally different types. In one the forces affect the lungs as a unit, whereas in the other the disturbance is concerned more especially with the mechanics of individual secondary lobules. In the non-industrial cases, involvement is of the general type, with bullae in the upper parts of the lungs; and in severe cases, with extensions or independent involvement of the interior parts. Gough[2] mentions that the bullae within the lungs are frequently distributed along the blood vessels, bronchi, septa, and in chains running up to the hilum. It is emphasized that they tend to occur in relation to all parts of the framework of the lung, whether in the interior or superficially along the pleura, suggesting that the absence of support for

the lung is not wholly responsible for the peripheral dilatations.

The distribution of bullae within the secondary lobules is the commonest type observed in bituminous coal workers; and a similar phenomenon is seen in non-industrial workers with severe chronic inflammation of the terminal bronchioles. While the bronchioles are not obstructed, it is suggested in Heppleston's studies, quoted by Gough[2], that an element of shrinkage of the coal foci is present, which, together with the force of inspiration, leads to over-stretching of the lung. Honeycomb lung or focal emphysema is associated with interstitial infiltration. With the former, the septa are usually involved, while in the focal type the process is mainly around the respiratory and terminal bronchioles.

Symptoms and Physical Signs

The symptoms of emphysema, occurring independently or in association with chronic pulmonary diseases, reflect to some extent the nature and extent of the pathology and the associated physiologic disturbances[3]. Cough, dyspnea, expectoration, and mental depression are of special significance. A "grunting" type of cough, precipitated during physical effort, occurs with respiratory embarrassment. In asthmatic states the cough is wheezy in type, suggesting the influence of bronchospasm. With superimposed infection, the cough is "loose" and usually associated with the accumulation of sputum. The expectorated material varies with the type of disturbance of the parenchyma and bronchial mucous membrane. For example, glairy, mucoid expectoration is common in tracheobronchitis and allergic states with emphysema; marked expectoration is present in bronchiectasis. With transient blockage of the bronchus and invasion of secondary organisms, there are lapses of expectoration followed by the discharge of foul material. Mental depression may be associated with long continued superimposed infection, whereby oxygen transfer from the lungs to cerebral tissues is impaired.

Dyspnea, or the consciousness of breathing during physical effort, is the most striking manifestation of emphysema. Accentuation occurs during episodes of chest colds and fever, and signifies bronchiolar blockage with bronchospasm. The severity of dyspnea should be carefully evaluated. As pointed out by Fletcher[4], the simple question, "How breathless are you on exertion?" will not suffice, for the answer will depend on the patient's age, habits, and physical activities. Since with advancing age tolerance of exercise is diminished, it is fallacious to compare the patient's present with his previous abilities. It is helpful to ask if the patient's breathing is as good as others of the same age and build on walking or on climbing stairs; also if he is unable to keep abreast with normal men on the level but is able to walk about a mile or more at a reduced speed. Breathlessness on talking or undressing is a feature of advanced emphysema.

The physical signs of emphysema in about 50 per cent of the cases are a barrel-shaped chest, widening of the intercostal spaces, obtuse angle of the ribs, elevation of the thorax occurring as a whole during inspiration, a preponderance of abdominal type of breathing, and clubbing of the fingers in cases with marked decrease in arterial oxygen[3]. In the remaining cases the thorax is elongated and flattened, and the respiratory excursions occur chiefly in the vertical plane, the abdominal movements being prominent during the respiratory cycle. With asthmatic states, the expiratory breath sounds are somewhat prolonged and harsh in quality; musical rales and wheezing sounds are not uncommon, especially in forceful breathing; indrawing of the interspaces is a feature of advanced cases. In other cases, especially those with pronounced fibrosis, the inspiratory phase is relatively prolonged, the rales are scattered, and the breath sounds distant[3].

Roentgen Signs

The roentgenograms of emphysema show increased illumination of the lung fields, with flattened, lowered, delayed and limited excursions of the diaphragm. The presence of abnormal shadows is an indication of acute or chronic processes, such as pneumonitis, atelectasis, fibrosis, and bulbous formations. Interestingly, the interpretation of the roentgenograms in some cases is debatable; for example, they may suggest the advanced condition and yet the patient may not be totally disabled; contrarily, the opposite relationship may exist.

Physical Dynamics

The patient's build, his habits of standing and sitting, the contour of the abdomen, and the behavior of the diaphragm may be

important factors in the evaluation, control, development, or perpetuation of pulmonary emphysema[5]. Impaired breathing, inadequate expectoration, unnecessary or ineffective cough, and failure to gain the proper values in respiration are sometimes traceable to mechanical disturbances associated with faulty physical dynamics. Thus, while abnormal mechanisms are contrasted with the normal, it may be suggested that the individual with improper thoracic and pulmonary dynamics is usually more conscious of his pulmonary symptoms than of his physical state and habits. Moreover, while the hypothesis that the onset of certain cases of emphysema may be related to hereditary factors, antecedent disease, and the wear and tear of life is accepted, it is apparent that the mechanical influences must be carefully considered.

The analysis of 59 persons with pulmonary emphysema suggested that impaired diaphragmatic and thoracic excursions are sometimes associated with the poorly developed or obese abdomen. Support for the hypothesis is the fact that air must be moved effectively in and out of the lungs; otherwise mixing and dilution problems will arise[5]. Actually, the efficiency with which the dead air "washes out" the alveolar air depends to a considerable extent on the amplitude of breathing.

In addition to the handicaps of faulty thoracic and diaphragmatic movements in respiration, there are the influences of a disturbed splanchnic area. According to Kerr[6], maintenance of the normal tone of the splanchnic blood vessels is essential to preventing the accumulation of blood in regions where it becomes static by the force of gravity. In debilitated persons in whom the splanchnic tone is diminished, as in ptotic individuals and those with obese and pendulous abdomens, it may be expected that a decrease in cardiac output and circulatory embarrassment will occur when the upright position is assumed. This is well illustrated by ordinary syncope, as in orthostatic hypotension and in attacks induced by standing still for a long time. The cause of this phenomenon is inadequate cerebral circulation induced by a loss of splanchnic tone.

Apparently this lack of compensation for postural changes also affects the coronary system, which in the presence of the pulmonary disease with inadequate oxygenation and gas exchange is disturbing to the general circulation. This aspect of abdominal tugging on the splanchnic area in a sense parallels the unfavorable effects of inadequate support from the abdomen, with faulty diaphragmatic action.

Physiology of Respiration

In normal breathing there is expansion of the chest with lowering of the diaphragm during inspiration, followed on expiration by elevation of the diaphragm and contraction of the thorax. The excursions of the lungs are both horizontal and vertical. Inspiration occurs as an active muscular effort, while expiration is essentially passive, depending on the recoil of the lungs and the elasticity of the chest wall. In strenuous exercise, the respiratory movements are modified, the diaphragm rising only partially during expiration, as the lungs continue in a state of distention. In a sense, expiration is imperfect, and each succeeding inspiration begins before expiration is fully accomplished.

The gaseous exchange between the alveoli and the pulmonary capillary blood vessels depends essentially on the physical process of diffusion; namely, on the difference of gas pressures in the alveolar air and blood. With these pressure differences, the solubility of the gas in the blood and the properties of the alveolar membrane are involved in the exchange of gases between the lungs and the blood. According to Wright[7], the pressure gradient in the case of carbon dioxide is one-tenth that of oxygen; the density of carbon dioxide is 44, compared with 32 for oxygen, but carbon dioxide is about 25 times as soluble as oxygen. It is suggested that carbon dioxide diffuses out into the alveoli more quickly than oxygen diffuses into the blood, in spite of disproportionate pressure gradients for the two gases[7].

The following physiologic aspects are especially significant in emphysema: The residual air percentage of total lung volume increases principally at the expense of the complemental air percentage of total lung volume; and the percentage portions of the lungs occupied by supplemental air (reserve) and tidal air remain nearly constant, regardless of the degree of emphysema. It is significant that with far advanced pulmonary emphysema, the breathing reserve may be markedly reduced, since the depth

of inspiration is increased only slightly above that of the resting state[8].

There also is faulty distribution of air in the lungs, resulting from the unequal aeration and perfusion caused by the fibrotic and obstructive changes in the lungs. Poor distribution of air lowers the arterial pressure of oxygen and percentage of oxygen saturation, owing to the drop in mean pressure of oxygen between the alveolus and the arterial blood. In some cases the increased mean gradient of pressure for oxygen between the alveoli and arterial blood approaches 50 mm. of mercury (normal, 5 mm. of mercury), and the average resting arterial oxygen saturation is moderately reduced but poorly correlated with impairment of pulmonary function. It is interesting that the arterial oxygen values obtained during mild exercise may reveal significant differences with respect to disability and the degree of emphysema, as illustrated when the arterial oxygen value is compared with the resting level.

Various tests are employed in emphysema for the evaluation of efficiency in gas exchange from the lungs to the atmosphere and from the lungs to the circulation. Vital capacity and maximal breathing capacity are the most important measurements of ventilation. Vital capacity decreases as the degree of emphysema increases. The test is useful, and yet the individual variations limit the value in certain cases; for example, there may be lowered vital capacity of 2,000 to 3,000 cc. with no manifest emphysema (determined by quantitative measurements of residual air), as well as in cases of moderate or even advanced emphysema. Maximal breathing capacity is related to the degree of emphysema in a manner similar to the correlation between vital capacity and emphysema. If the maximal breathing capacity is less than 40 liters per minute, significant emphysema is present (residual air 35 per cent or more of total lung volume); and if the maximal breathing capacity is over 100 liters per minute, the impairment, in most cases, is not due to emphysema.

Comparisons between step-up and treadmill exercise, using arterial blood and respiratory gas exchange measurements, provide important information[9]. In 17 patients the treadmill exercise produced a slight decrease in arterial oxygen saturation, and in 13 patients a significant increase of oxygen saturation (3 per cent plus). The data also indicate that the pulse and respiratory rates, blood pressure, and percentage of recovered oxygen do not vary significantly between the two methods of exercise, but in 36 patients with an important degree of emphysema (residual air over 35 per cent of total lung volume) the arterial oxygen saturation showed a more striking decrease during step-up exercise.

Treatment

The treatment of emphysema is concerned with the prevention or control of excessive cough and chronic pulmonary diseases. The further aspects relate to the physiologic rehabilitation of reversible disease, as follows: (1) measures favoring normal gas exchange and the removal of abnormal secretions from the air cells and bronchial passages; and (2) procedures that will encourage effective breathing, both in the mechanical and physiologic aspects. Preservation of the whole dynamic action of the respiratory system encourages adequate ventilation and gas exchange[8].

Medical direction should include the correction of metabolic states, especially obesity, since excessive weight is a burden on the failing pulmonary circulation. It is desirable also to eliminate chronic infections of the teeth and respiratory passages to avoid the gravitation of pus and abnormal secretions into the dependent parts of the lungs. Sinus disease and allergic states will accentuate and sometimes accelerate the course of fibrosis, atelectatic processes and emphysema, and with their first appearance it becomes essential to avoid dust hazards, dampness, and extreme cold. Tobacco smoking may or may not be harmful, and its avoidance should depend on individual susceptibility. Heart disease and hypertension predispose to pulmonary disability, and with these conditions arduous work is contraindicated. Rest in bed, as a therapeutic measure, requires careful regulation because of its retarding influences on bronchiolar drainage and pulmonary ventilation. It should be emphasized that excessive use of the rocking chair is detrimental, as is physical exertion in active tuberculosis[8]. Gymnastics, with deep breathing exercises on rising from bed in the morning, are valuable as a means of improving the general condition of the lungs and certainly in favoring adequate ventilation.

Graduated activities during the day are also valuable, but fatigue, chest discomfort, increased pulse or respiratory rate call for limitations; stair climbing and rapid walking, especially against the wind, should be avoided[8]. Postural drainage, obtained by elevating the foot of the bed, is indicated for basal bronchiectasis. With careful trial it is possible to determine the most satisfactory position for drainage; for example, rolling over periodically from side to side is effective in stimulating the flow of expectoration Resting on the abdomen will also encourage diaphragmatic function, as the stomach, intestines and other organs are shifted upward.

The use of drugs alone in emphysema and the complicating chronic pulmonary diseases has been disappointing, often harmful[3]. The expectorant and sedative drugs are more disturbing than effectual; codeine and other morphine derivatives retard respiration and favor the retention of secretions; codeine is indicated only for severe, racking cough and hemorrhage; morphine should be given only for severe pain. In failure of the circulation due to primary disease of the myocardium, digitalis, theobromine derivatives, and mercuhydrin are valuable, especially with oxygen therapy. Terramycin, in adequate dosage (250 mg. every 6 hours), may be given parenterally for episodes of acute respiratory tract infection; penicillin, given parenterally in doses of 500,000 units once or twice daily, is valuable for excessive expectoration and bouts of acute bronchial infection in associated bronchiectasis. Cortisone may provide symptomatic relief in dyspnea, but the effects are temporary and sometimes harmful in the presence of acute pulmonary diseases, especially tuberculosis. Surgical removal of cysts which harbor dead air and compress normal tissue should be done in cases with favorable pulmonary function.

Physicophysiologic Methods in Treatment

The use of physicophysiologic methods in treatment requires a careful study of the abdominal and diaphragmatic influences, the goal being to utilize their favorable action for improving bronchiolar drainage, alveolar ventilation, and the transfer of oxygen to the tissues. Faulty habits of sitting, standing, and walking should be corrected. As a first step in the program, relaxation of the shoulders, arms, and thorax should be cultivated in order to avoid overstretching the pulmonary tissues and to encourage equalized chest movements. While the abdomen and diaphragm take over the preponderant role of lung ventilation, the effects of relaxed breathing will be simplified. The practice of "standing at attention" with the shoulders squared, the neck stiff and hands on the hips, as well as of sitting with the elbows supported on the arms of a chair should be avoided. For a lesson in relaxation and efficient breathing, patients should watch ball players as they run out on the field; and for the proper sitting posture, they should experience the comfort and ease of breathing while resting in a straight chair[10].

Adequate diaphragmatic elevation with suport for the abdomen improves pulmonary ventilation and blood flow to the heart. This may be accomplished mechanically by a device consisting of a pad, back piece, and two cross-springs of curved light-gauge steel, each 12 inches in length[11]. With this arrangement, "through and through" pressure is attained, giving to the abdomen a lifting type of support as well as compression; strong pressure is not required. Patients are taught to "settle" down into the appliance so as to gain the maximum benefit. The physiologic action results from "lifting" the abdomen, thus reducing "tug" on the splanchnic area while shifting the diaphragm to a higher range of operation.

In supplementing the effects of abdominal elevation and compression, a form of respiratory exercise is usually required, especially for patients with persistent dyspnea, racking cough, wheezing, and difficult expectoration due to bronchospasm. The procedure currently recommended is intermittent positive pressure breathing (oxygen, 100 per cent) with vaponefrin (6 drops diluted in the same amount of distilled water) employed simultaneously as an aerosol[12]. Alevaire, a preparation with a detergent-like action, may be added to the vaponefrin solution in cases with thick tenacious mucus; the usual dose is 1 to 2 cc. The apparatus is essentially a cycling valve (flow sensitive) with a mouthpiece, nebulizer, gauges, regulators, and other units arranged conveniently for the introduction of oxygen and aerosol into the respiratory passages. Treatments are given two or three times daily (5 to 10 minutes each) for about three months or until relief is obtained; then two or three times weekly.

The sustained benefits of treatment with

intermittent positive pressure breathing are variable, as would be expected in aging persons with severe pulmonary manifestations. In some instances relief is gradually increased over a period of three to four months, while in others it is only transitory between treatments. Generally the improvement is sufficient to permit a more active and enjoyable life. The features include lessened dyspnea, fatigue, and mental depression, with improved digestion and gain in weight.

Summary

The clinical and physiologic aspects of emphysema are related to a variety of factors, including weakened, thinned and dilated alveoli, bronchospasm, bronchiolar infection, the general effects of chronic pulmonary diseases, and faulty posture. Accordingly, the signs and symptoms are somewhat proportionate to the fundamental disease and associated conditions.

The concept that oxygen lack is the chief cause of the symptoms of emphysema—notably dyspnea, fatigue, insomnia, nervousness, and depression — is supported by observations following the use of physicophysiologic dynamics. Intermittent positive pressure breathing (oxygen) treatments, a form of respiratory exercise, provides a more uniform aeration of those alveoli which are poorly ventilated on ambient breathing but are still perfused with blood. When the treatments are supplemented with the use of a special abdominal support to provide a compressing and lifting effect on the abdomen, the relief is sometimes increased. Correctional methods for improving postural defects and faulty habits of breathing are effective in gaining a more adequate alveolar aeration. Dynamic treatment is proposed as a means of rehabilitating groups of impaired alveoli and bronchioles, thus improving the function of the lungs.

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ANESTHESIA FOR SURGERY OF THE HEART AND GREAT VESSELS

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The development of operative procedures for the treatment of congenital and acquired heart disease has been a brilliant and exciting chapter in surgical progress of the last decade. This has been the impetus for the development of new concepts of anesthetic management. The purpose of this paper is to show the correlation between the anesthetic management of operations on the heart and great vessels, and the abnormal hemodynamic alterations associated with congenital and rheumatic heart disease.

The provision of adequate oxygenation, the elimination of carbon dioxide, and the establishment of a stable mediastinum are presupposed fundamental requirements for all intrathoracic procedures. Associated with these prerequisites special considerations are necessary in the anesthetic management of operations for patent ductus arteriosus, tetralogy of Fallot, coarctation of the aorta, and rheumatic heart disease with mitral stenosis.

Patent Ductus Arteriosus

Pathologic physiology

The ductus arteriosus, which connects the aorta and the pulmonary artery during fetal life, usually closes within four months after birth. Persistent patency provides a channel for the recirculation of oxygenated blood

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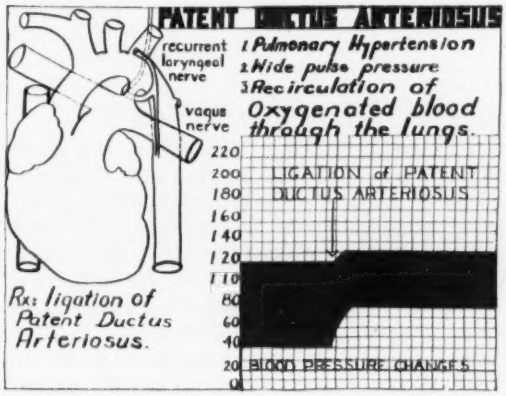

Figure 1.

to the lungs. The hemodynamic effects are increased blood flow through the pulmonary artery, pulmonary hypertension, and decreased peripheral resistance. The latter is manifested by a low diastolic pressure and a wide pulse pressure. More than half of the left ventricular output may be shunted back into the pulmonary circulation. To compensate for this inefficiency, the left ventricular output increases by increasing the cardiac rate and stroke volume. This increase in the work of the left ventricle is manifested by a rapid, actively pounding heart and eventually by left ventricular hypertrophy. The operation is designed to obliterate the arteriovenous shunt and thus restore normal hemodynamics.

Premedication

Adequate premedication is essential for a quiet, smooth induction. Morphine sulfate is given in accordance with the age, weight, and degree of the individual's reflex irritability. The dose of atropine sulfate is reduced so as not to enhance the pre-existing compensatory tachycardia. The dose of atropine is 0.05 mg. per 10 pounds of body weight.

Anesthetic management

The anesthetic technique has been the closed to-and-fro or circle carbon dioxide absorption endotracheal method. The anesthetic agent has been cyclopropane for induction, with either cyclopropane, ether, or both, for maintenance.

Extreme caution should be used in patients with high pulmonary artery pressures or frank reversal of flow. Agents which produce peripheral vasodilatation with a decrease in the peripheral resistance will initiate or enhance a reversal of flow, with subsequent hypoxia and cyanosis.

The respirations are compensated to insure efficient pulmonary ventilation and a stable mediastinum. During the course of dissection, the left vagus nerve may be stimulated, with resulting bradycardia, hypotension, and arrhythmia. If this occurs, surgical manipulation should cease and not be resumed until the pre-existing cardiac rate and blood pressure are restored. The closure of the ductus arteriosus is followed promptly by a rise in the diastolic pressure and a narrowing of the pulse pressure. This is a manifestation of the increased peripheral resistance. The cardiac action becomes quiet, less forceful, and more efficient. A decrease in the pulse rate has been observed in some cases. The pulmonary pressure returns to normal.

Supportive therapy

The average patient suffers minimal blood loss during the operation. These patients consistently have been shown to have an increased blood volume before operation. The closure of the ductus arteriosus by restoring the peripheral resistance toward normal may impose an increased circulatory load upon the heart. This adjustment should not be further disturbed by an excessive circulating blood volume. If blood replacement is indicated, it can be safely expedited following the obliteration of the ductus.

Tetralogy of Fallot

Pathologic physiology

As originally described in 1888 by Fallot, the essential features of the tetrad are pulmonary stenosis or atresia combined with dextroposition of the aorta, a high ventricular septal defect, and right ventricular hypertrophy. The primary hemodynamic alteration in the combination of defects is a reduction in the effective pulmonary blood flow, resulting in an inadequate oxygen uptake to meet the metabolic demands. Other hemodynamic derangements are pulmonary hypotension, narrow pulse pressure, and a recirculation of unoxygenated blood to the tissues.

These patients with chronic hypoxemia develop certain compensatory mechanisms in an attempt to overcome hypoxemia. The most important of these compensations is the development of polycythemia. Polycy-

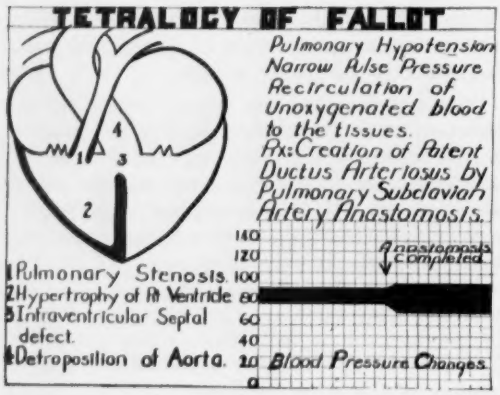

Figure 2.

themia, although of initial benefit in increasing the oxygen uptake of the arterial blood, often becomes excessive and potentially hazardous. Overcompensation results in increased blood viscosity, with subsequent slowing of the peripheral blood flow. The peripheral blood flow associated with increased utilization of oxygen is manifested clinically by cyanosis. The increased blood viscosity predisposes to thrombosis and increases cardiac work, with subsequent severe strain upon the myocardium.

The corrective operation for this cardiac anomaly is designed to increase the circulation to the lungs and enhance the uptake of oxygen. The hemodynamic alterations are almost exactly the reverse of those associated with patent ductus arteriosus. It is logical to assume that the creation of an artificial ductus arteriosus would be beneficial. This is accomplished by the Blalock-Taussig technique of pulmonary-subclavian artery anastomosis or, more rarely, by the Potts-Smith technique of pulmonary-aortic anastomosis.

Premedication

Psychic sedation and reduction in metabolic activity assumes increased importance in these patients with extreme hypoxia and low cardiac reserve. Excitement, fear, or crying may produce unconsciousness or convulsions, owing to the patient's inability to adjust to an increased oxygen demand. Morphine sulfate (1 mg. per 10 pounds of body weight) and atropine sulfate (0.05 mg. per 10 pounds of body weight) is given for premedication. The large dose of morphine adequately reduces the metabolic activity and increased oxygen demand. No undue respiratory depression has resulted from this dose of morphine. This combination of premedicants produces a quiet, drowsy patient and results in a smooth induction, without struggling or excitement.

Anesthetic management

The special problems involved in the anesthetic management of patients with tetralogy of Fallot usually are the direct result of altered hemodynamics. The use of generalized hypothermia to reduce cellular metabolism and oxygen demand during the operation appears to be a sound physiologic procedure in these hypoxic patients. Following further animal studies, its use is contemplated in the near future. The anesthetic technique in all of these cases has been the closed to-and-fro carbon dioxide absorption endotracheal method. Cyclopropane has been the anesthetic agent of choice, because it facilitates a rapid and smooth induction with high oxygen concentrations, and produces a quiet operative field undisturbed by excessive respiratory movements. With this agent, the depth of anesthesia can be adjusted rapidly.

The color of most of the patients improved following premedication, and further improved when general anesthesia was established. This was attributed to the decrease in metabolic activity and oxygen demand. The reduced pulmonary blood flow with hypoplasia of the pulmonary vessels and the relatively bloodless state of the lungs increases the problem of administering the anesthetic gas and oxygen. The respirations are compensated throughout the operation except during the period of the anastomosis, at which time they are controlled. The very delicate procedure of performing the anastomosis is facilitated by the relative lack of movement in the operative field produced by controlled respirations. The operation has usually consisted of an end-to-end anastomosis of the subclavian and pulmonary artery, so that both lungs receive an increased flow of blood.

The problems of anesthetic management become more difficult at the time of the anastomosis. An average of 38 minutes has been required to accomplish the procedure, which calls forth an exaggerated respiratory effort, difficult to control. This is due to the strong respiratory drive of the aortic and carotid chemoreceptors resulting from the increased hypoxic stimulus. The exaggerated respiratory effort ceases as soon as the new ductus arteriosus is opened and the blood

becomes better oxygenated. Deepening the plane of anesthesia usually facilitates the accomplishment of controlled respirations. On three occasions curare proved to be necessary, despite reluctance to use this agent for fear of masking the warning signs of asphyxia.

The development of bradycardia is felt to be of vagal origin in the presence of hypoxia. It is best treated by increased oxygenation and the administration of atropine.

Opening the anastomosis increases the pulmonary blood flow, the pulse pressure, and the work of the left side of the heart. Surprising though it may be, pulmonary edema or cardiac failure has not occurred in our series to date. Death during the operative day has occurred as the result of the unavoidable interruption of collateral circulation incident to thoracotomy and mediastinal dissection, which reduces an already decreased arterial oxygen saturation to dangerously low levels. Decision not to attempt the anastomosis because of the lack of suitable vessels results in a mortality rate of over 50 per cent.

Supportive therapy

A cannula for the continuous administration of fluids and supportive therapy is always inserted into the saphenous vein at the ankles the night before operation. Twelve hours of dehydration before operation increases the danger of hemoconcentration and thrombosis. Although whole blood and plasma are always available during the operation, they are rarely used except when blood loss is felt to be excessive. These patients have such a small cardiac reserve that care must be taken not to overload the circulation.

Coarctation of the Aorta

Pathologic physiology

The anomaly of coarctation of the aorta consists of a constriction of a segment of the aorta in the region of the ligamentum arteriosum. The hemodynamic alterations associated with this anatomic defect consist of hypertension in the upper extremities, hypotension in the lower extremities, and the development of a collateral circulation from the region above the constriction of the aorta to the region below. The operation is designed to resect the coarctation and establish the normal anatomy by end-to-end anastomosis.

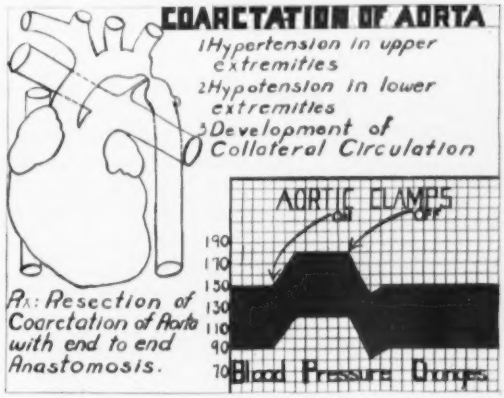

Figure 3.

Premedication

The pre-anesthetic medication consists of a combination of morphine and atropine, and is given in the usual doses, depending upon age, weight, and degree of the individual's reflex irritability.

Anesthetic management

The anesthetic agent is ether or cyclopropane administered by the closed endotracheal circle or to-and-fro carbon dioxide absorption technique. The special problems involved in the anesthetic management of patients with coarctation of the aorta arise when the aortic clamps are applied and released. The excessive hypertension following application of the clamps has been reported on occasions to produce reflex bradycardia and convulsions. The most dangerous period of the operation occurs when the clamps are released. Sudden cardiac arrest has been experienced in many university hospitals at this critical period, usually occurring shortly after the clamps are removed. The sudden fall in blood pressure and subsequent cardiac asystole is due to too rapid removal of the clamps. This sudden change in the vascular bed is too great for the usual compensatory mechanisms to readjust. The rapid fall in blood pressure decreases coronary blood flow, producing severe myocardial ischemia. The degree of hypertension and the relative rather than the actual fall in blood pressure are important factors in the development of cardiac arrest. The clamps should be removed slowly over a period of time that is determined by the stability of the blood pressure. This will allow the patient's compensa-

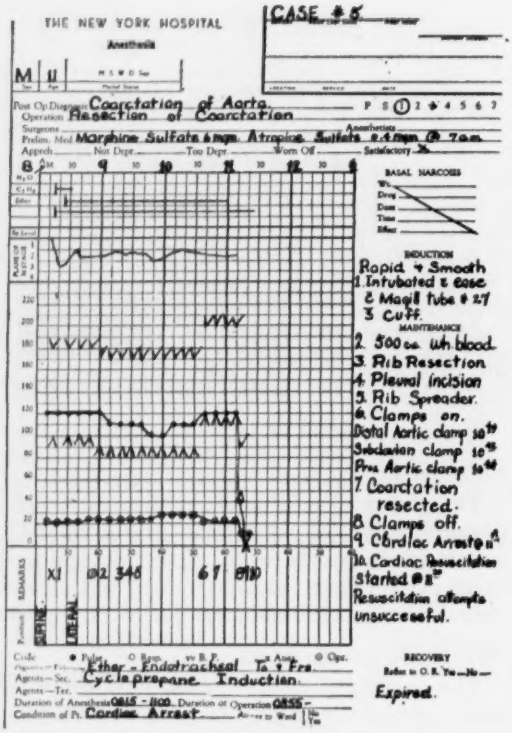

Figure 4.

tory mechanisms to make the necessary cardiovascular adjustment.

There has been only one mortality in our series of surgical correction for coarctation of the aorta. The patient was an 11 year old white boy who was induced with cyclopropane and maintained on ether administered by the endotracheal to-and-fro absorption technique. The operative course was uneventful until the aortic clamps were removed, when cardiac arrest occurred simultaneously. All methods of cardiac resuscitation were unsuccessful.

Supportive therapy

Cannulas are inserted in the saphenous vein of each ankle to provide a means of rapid blood administration when indicated. Because of the extensive collateral circulation, the opening of the chest may entail considerable blood loss. It is important to replace only the blood lost before the clamps are applied. This will prevent the increase in blood pressure that would result from the hypervolemia. This amount of blood replacement will tend to insure an adequate blood volume at the time the aortic clamps are removed.

Rheumatic Heart Disease with Mitral Stenosis

Pathologic physiology

Anesthesia for the patient with rheumatic heart disease who is chosen as a candidate for mitral valvulotomy demands an individualized regimen. The pulmonary hemodynamic derangements resulting from the mechanical effect of the narrowed mitral orifice well illustrate this fact. The subsequent elevation of the left atrial pressure results in the development of hypertension in all segments of the pulmonary vascular bed—venous, capillary, and arterial. The cardiac output is reduced and relatively fixed. The pulmonary vascular resistance is invariably increased, adding to the work load of the right ventricle. The cause of the increased pulmonary vascular resistance is not completely understood, although physiologic as well as anatomic alterations in the arterioles are usually present. Pathologic studies of the lungs have revealed varying degrees of arteriolar sclerosis, thickening of the basement membrane of the alveolar capillaries, and pericapillary edema and fibrosis. The considerable reduction in the pulmonary vascular resistance that follows mitral valvulotomy suggests that physiologic factors are partially responsible for its elevation. The hydrostatic pressure in the pulmonary capillaries frequently approximates the plasma colloid osmotic pressure. The ease with which pulmonary edema may develop in this setting of altered pulmonary hemodynamics is apparent.

Improvement in the pulmonary hemodynamics follows mitral valvulotomy in the ideal candidate. This is manifested by an increased cardiac output and a reduction in the pressures in all segments of the pulmonary vascular bed.

Premedication

The premedication has consisted of a combination of morphine and atropine. Morphine sulfate is given in small doses not to exceed 8 mg. for fear of producing respiratory depression with resultant hypoxia and increase in pulmonary arterial pressure. Atropine has been used in small doses not exceeding 0.2 mg. for fear of a moderate degree of vagal blockade. An increased cardiac rate allows less time for diastolic filling, and

hence stroke volume is decreased. This may initiate cardiac decompensation. Disturbing episodes of sinus tachycardia have not been observed with this dose of atropine. This is attributed to adequate preoperative digitalization.

Anesthetic management

Ether has been the primary agent, using the endotracheal closed circle carbon dioxide absorption technique. To facilitate a smooth induction, 2.5 per cent thiopental sodium has been given until the patient no longer responds to spoken voice. This in all cases has been an extremely small dose, not exceeding 200 mg. A 50 per cent mixture of nitrous oxide and oxygen is used to accomplish the thiopental-ether sequence.

The depth of anesthesia is more important than the agent or technique. Ether produces peripheral vasodilatation in proportion to the depth. Because of the low cardiac reserve and relatively fixed cardiac output, these patients are unable to compensate for this peripheral vasodilatation, and a subsequent peripherovascular collapse develops. Intubation is accomplished in the first plane of the third stage, aided by topical spray of the larynx with 2 per cent xylocaine and the application to the tube of 5 per cent xylocaine ointment to minimize a bucking response and the subsequent hypoxia that may develop with this reflex. The patient is then maintained in the top of the first plane of the third stage of general anesthesia.

In a series of 70 cases, no patient has received more than 1 to 1½ ounces of ether for the entire induction and maintenance. Relaxant drugs have not been found necessary. The regional intercostal nerves are blocked with procaine as the chest is opened. This prevents some of the cardiorespiratory irregularities that occur with rib-spreading. When the chest is open, the respirations are compensated to insure efficient pulmonary ventilation and a stable mediastinum Extreme care should be exercised in the use of compensated respiration for fear of producing myocardial overdose with ether. Controlled respiration should not be used for fear again of the likelihood of myocardial overdose. There is also grave danger of impeding blood flow in the pulmonary system, which is usually at borderline compensation in this group of patients. At the time of palpation of the auricular appendage for clots and the insertion of the finger through the valve, bilateral compression of the carotid arteries at the neck is applied in an attempt to prevent cerebral embolization.

The Cambridge Cardioscope at the side of the anesthesiologist has been of inestimable value in following the cardiac rate and rhythm in these cases. The continuous electrocardiogram allows the anesthesiologist to inform the surgeon when he is working in areas of high irritability. These areas are most frequently associated with pressure in the vicinity of the descending branch of the left coronary artery.

In the early cases of our series, procaine was used as a continuous intravenous drip and applied topically on the myocardium as a prophylactic measure during surgery. It soon became apparent that the depressant effect of this agent on the ventricular muscle was of greater disadvantage than the advantage of preventing ventricular arrhythmias. Irritability and conductivity are normal properties of the myocardium, and should not be depressed with procaine. Therefore, procaine in all its forms has been eliminated for routine use, and is reserved only for specific indications. The greatest number of cardiac irregularities occur during the manipulation of the mitral valve. A few ventricular extrasystoles or even a short run of ventricular tachycardia are most commonly seen. This is probably due to distortion of the extremely sensitive upper ventricular septum by the finger or knife during the manipulation. It is quite comforting to know that in the overwhelming majority of instances, as soon as the finger is held stationary in the auricle there is a quick return to the preexisting rhythm of the heart. At present, no attempt is made to stop the surgeon from manipulating the valve unless ventricular tachycardia persists for 30 seconds or more, or a persistent bizarre rhythm occurs.

Supportive therapy

The administration of whole blood must be carefully controlled so as to replace only that blood which is lost. Circulatory overload occurs easily in these patients. Conversely, a decreased blood volume may seriously incapacitate this damaged heart.

The high incidence of severe postoperative chest pain which these patients formerly experienced is now controlled by blocking the intercostal nerves with Efocaine. The intercostal block should not be done in the paravertebral region for fear the solution

will leak into the subarachnoid space through a prolonged dural cuff and cause the development of a severe cord lesion. Despite relief of this intercostal pain, there may develop in a period of weeks or months a substernal pain which may radiate to the shoulder and is aggravated by swallowing and deep breathing. Phrenic block has been partially effective in treating this type of pain, which is most likely pericardial in origin.

Summary

Special problems involved in the anesthetic management of operations on the heart and great vessels of patients with congenital and rheumatic heart disease have been discussed. These special problems are usually the direct result of altered hemodynamics. A thorough understanding of the underlying anatomy, pathologic physiology, and resultant hemodynamic derangements is imperative for the proper anesthetic management of these patients.

Discussion

Dr. C. R. Stephen (Durham): I would like to congratulate Dr. Crandell on his comprehensive paper and the excellent work that is being done in cardiovascular surgery at Cornell University. The point of emphasis is that anesthetists are trying to understand the underlying physiology, normal or abnormal, in patients presented for operation. We have felt for some time that probably the most difficult type of surgical anesthesia with which one is confronted is that for the correction of tetralogy of Fallot. As has been pointed out, these patients are extremely cyanotic, their blood viscosity is very high, and during the course of the anesthesia they constantly present problems which can be quite difficult to overcome.

We feel also that perhaps the method of administering the anesthesia is more important than the agent. Cyclopropane is satisfactory in these cases because it is readily reversible and the depth of anesthesia can be varied with relative ease. Perhaps the most important object, however, is to maintain as adequate oxygenation and elimination of carbon dioxide as one can under the circumstances. This means, of course, complete and continuous control of respiratory exchange. It is not enough to see that the breathing bag is moving; it must be moving in such a way as to indicate that the exchange—or shall we say the tidal volume of the patient?—with each respiration is equal to what one would expect if the patient were wide awake.

The anesthetist's great warning sign in the tetralogy of Fallot is the increasing bradycardia that some of these patients show. As Dr. Crandell has pointed out, the incidence or the gradual progression of bradycardia means, probably, that the patient is suffering from anoxia. Sometimes we can't do anything about that condition, since it may be cardiovascular in origin rather than respiratory, but in other cases, we can. As Dr. Crandell points out, the administration of atropine is extremely important. If the heart responds by beating more rapidly and the bradycardia is overcome, we can do something for the patient.

I remember vividly one patient who, when the pulmonary artery was clamped off, began to show bradycardia and gradually went downhill, the pulse becoming slower. Atropine, vasopressors were given —everything we knew—in an effort to increase the pulse rate. Though we felt that the oxygenation was entirely adequate, nevertheless this patient died in front of our eyes. At autopsy we found there was no pulmonary artery on the other lung, indicating that there had been sufficient circulation through the bronchial arteries to supply adequate oxygenation.

Under normal circumstances, the incidence of bradycardia primarily means anoxia. If we can overcome that lack, and if by drugs we can counteract the vagal stimulus which may be produced in the bradycardia, we probably are doing the best we can under the circumstances.

THE PREVENTION OF CARDIAC ARREST DURING ANESTHESIA

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Much attention has been directed during recent years to the dangers of cardiac standstill or ventricular fibrillation during or immediately following anesthesia. Emphasis has been placed on the early recognition of the emergency and on heroic treatment by cardiac message and the use of an electric defibrillator, if needed. The dramatic recovery of some patients has led to much publicity in the lay press, as a result of which some physicians have warned that failure to have available or to use an electric defibrillator when indicated would constitute gross negligence and malpractice. Some fear, however, that such emphasis might lead to unjustified attempts at cardiac massage on patients with sudden coronary occlusion or Stokes-Adams attacks who might otherwise survive with approved medical therapy[1].

In 350 collected cases of cardiac arrest recently reported[2], there was an over-all mortality of 65 per cent. In the most favorable report, in 47 cases in which immediate and adequate cardiac massage was instituted, the mortality was still 15 per cent. Certainly, more emphasis should be placed on the prevention rather than on the treatment of cardiac arrest. This requires a review of the fundamental factors at work in maintaining a regular and effective heartbeat.

Many such factors are involved, creating a state of confusion which is well illustrated

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by the following quotation from Dinsmore[3], in a discussion of a paper on cardiac arrest.

> "First of all, the patient is met in the operating room by a man with a scalpel and another fellow with a (gas) machine with everything on it but a sledge hammer . . . The patient may be given some multiple barbiturates early in the morning, followed by morphine, then by atropine, and then a little bit of Pentothal for induction, and actually it is easier to place an intratracheal tube in if one uses a little cocaine. Then one gives the patient some nitrous oxide, some ether, or some cyclopropane . . . Then you start to operate and the abdominal wall is rigid, and the surgeon becomes rigid, and the anesthetist limp; then you get some curare. You cannot tell me that all those drugs and agents are not factors (in cardiac arrest)."

This point of view is shared by many surgeons. The surgeon, being more conscious of the possibility of cardiac arrest, is wondering if the anesthetist is aware of the possible cumulative effects of these anesthetic agents, if he is alert to note and correct immediately any deviation from normal heart action, and if he is competent to recognize danger signals and to demand that the surgeon allow him to get the patient stabilized before proceeding further. The basic question the surgeon would ask of the anesthetist is: Are you familiar with the fundamental physiology of the heart's action, and are you, during every anesthetic procedure, as concerned with maintaining this normal action as you are with maintaining anesthesia and muscular relaxation?

Factors Affecting the Action of the Heart

The fundamental factors affecting the integrity of the heart's action can be reviewed, those which are especially liable to be altered during anesthesia can be emphasized, and practical measures to prevent or correct these alterations can be suggested. Anything which threatens the functional integrity of either the heart muscle, the conduction tissues, or the coronary circulation can impair cardiac action and ultimately lead to cardiac arrest.

The known factors which may alter this integrity during anesthesia may be grouped as nervous influences, chemical influences, anesthetic agents, and surgical shock from hemorrhage.

Nervous Factors

Nervous influences affect principally the heart rate[4]. Basically, the parasympathetic or vagus nerve supply tends to decrease the heart rate, while the sympathetic supply tends to increase it. Actually, the cardiac accelerator and cardiac inhibitor centers in the brain are reciprocally in tune, and each exerts a continuous although varying tone on this pacemaker mechanism. The predominant tone at any one time is influenced by the reflexes active at that time.

It is well known that vagus stimulation leads to bradycardia by directly affecting the sino-auricular node, the auricles, and the AV bundle. Many reflexes which can lead to this vagal effect and even to temporary cardiac standstill have been recognized. Among them is irritation of the respiratory or gastrointestinal tract, such as occurs with insertion of an endotracheal tube, a bronchoscope, gastroscope, nasal suction catheter, or nasal pack[5]. Vagal stimulation can also constrict the coronary vessels, decreasing the blood flow through them. Predominantly, sympathetic reflexes lead to tachycardia not only of the auricles but also of the ventricles by increasing the excitability of the conduction tissues and of the muscle itself. One of the strongest reflexes of this nature is that due to hypoxia[4]. Stimulation of a peripheral nerve such as occurs in amputations or with operations upon the ribs can lead to a similar effect.

An important point to remember in regard to these nervous reflexes is that during a general anesthesia the sympathetic reflexes are depressed sooner than are the vagal[6]. In the stage of surgical anesthesia, therefore, one would expect vagal tones to predominate. It is for this reason that atropine is so useful, not only to prevent excessive bronchial secretions, but also to guard against undue depression of the heart rate. On the other hand, during induction the sympathetic reflexes are still active, and may be stimulated by fear and excitement and augmented by a concomitant outpouring of adrenaline[4]. One would expect rapid heart action to predominate, if poor respiratory exchange and resulting hypoxia are added to the picture. Here certainly the art as well as the science of anesthesia is necessary for smooth induction. Adequate premedication must be supplemented by a confident manner and good rapport with the patient.

Chemical Factors

Hypoxia

Chemical influences upon the heart are mediated not only through the nervous reflexes, but directly on the heart muscles and

conduction system through the blood. The dangers of hypoxia are numerous. The heart muscle itself cannot beat longer than 10 minutes without oxygen. It cannot take an oxygen debt as skeletal muscle can. When so deprived of oxygen, it becomes more excitable, as evidenced by numerous extrasystoles and even heart block[4]. The poor muscular contraction and dilatation lead to poor output with further decrease of coronary flow and a further degree of hypoxia, thus instituting a vicious cycle which can terminate suddenly in cardiac arrest.

Added to this direct effect on the muscle itself are the reflexes which go into play with generalized hypoxia. Since vagal tone may predominate to depress the sinus rhythm, the hyperirritable heart muscle and conduction tissue may set up ectopic pacemakers with gross arrhythmias[4]. The increased adrenaline in the blood only aggravates the picture by trying to accelerate an already excitable heart.

The factors which may lead to hypoxia are legion and for the most part are well known. All anesthetic agents, but especially cyclopropane and the barbiturates[7], can cause respiratory depression. In addition, any obstruction to the airway by excess mucus or spasm or too much curare or an unfavorable position on the table can lead to hypoxia. The high Trendelenburg position may limit motion of the diaphragm, the lateral kidney position may prevent adequate exchange. It is well known that the open chest leads to poor ventilation, especially in the lateral position[8].

Hypercapnia

Hypercapnia, or an accumulation of carbon dioxide, results in a lowering of blood *p*H, which in itself depresses AV conduction and relaxes the heart muscle[4]. In one study[8d] one patient was found to have a *p*H of only 7.2 from the premedication alone. Carbon dioxide is itself a narcotic and anesthetic. This has been demonstrated frequently during general anesthesia when the oxygen content of the inspired air and of the blood was still within normal range[8a]. The heart is much more vulnerable to the effect of vagal stimulation when the carbon dioxide content is elevated and the blood *p*H depressed[9].

The danger of carbon dioxide accumulation is much greater than that of hypoxia because it cannot be readily recognized. One can look at a cyanotic patient and know that he needs oxygen, but there is no adequate way as yet to determine rapidly the need for better removal of carbon dioxide. It is wise to assume that any cyanotic patient not only has too little oxygen but also has too much carbon dioxide. This situation cannot be corrected solely by increasing the concentration of oxygen in the respiratory mixture. This only gives the patient a pink color without removing the carbon dioxide, and the anesthetist is unaware of the defect which should be corrected. Waters[10] has emphatically stated that oxygen should be delivered in a normal concentration of 20 per cent; then if the patient becomes cyanotic, he either has respiratory depression, respiratory obstruction, or both. The cyanosis is a warning sign, demanding immediate action to clear the airway and assist respirations rather than to increase the oxygen concentration and obscure the whole picture.

Another danger of carbon dioxide accumulation is its effect on the vasomotor center, which raises the blood pressure[4,7a]. This is one clinical sign of value. However, this sign in itself may temporarily mask or be masked by the effect of blood loss which has been inadequately replaced. As a consequence, surgical shock and hypoxia, with their effect upon the heart, are more prone to occur when the carbon dioxide is blown off at the conclusion of the procedure[7a]. The surgeon as well as the anesthetist must beware of this effect of carbon dioxide upon the blood pressure. The blood pressure should not be used as the sole gauge for adequate blood replacement, but should be supplemented by a realistic estimate or actual measurement of the loss. There is definite evidence[11], furthermore, that the sudden reduction of high carbon dioxide tensions causes not only hypotension but also ventricular arrhythmias and fibrillation.

Asphyxia results when hypoxia and accumulation of carbon dioxide occur simultaneously. The effects of each are enhanced, and the reflex effect of vagal stimulation is even more pronounced[9].

Other factors

Other chemical factors which might affect the heart action may be summarized briefly. Accumulation of lactic acid as a result of hypoxia or metabolic acidosis with some agents has an effect similar to that of carbon dioxide by lowering the blood *p*H. An

elevated calcium tends to increase the contractility of the heart and will eventually lead to calcium rigor. Although there is no definite information available, it is suggested that a lower *p*H from any of the above causes might lead to greater ionization of calcium and adversely affect the heart. Blood potassium works reciprocally with calcium, leading, if elevated, to cardiac inhibition, which should be guarded against in patients with uremia. On the other hand, a low potassium may act as an increase in calcium and can occur with dehydration from excessive loss of fluids and electrolytes in the preoperative period.

Anesthetic Agents

All anesthetic agents can lead to some respiratory depression, with concomitant hypoxia and hypercapnia. Actually, cardiac arrest has been reported during all forms of anesthesia, including local and spinal[2]. Many[10] agree that cyclopropane *per se* definitely causes arrhythmias as well as sensitization of the heart muscle to the action of circulating adrenaline, which can lead to ventricular tachycardia. It seems obvious that when the respiratory exchange has been compromised by any anesthetic agent, respirations must be assisted; and when cyclopropane is used and an arrhythmia occurs, this agent should be removed immediately.

Hemorrhage and Shock

Hemorrhage and surgical shock affect the heart through the resulting hypoxia and lowering of blood pressure and coronary flow. The danger that this factor will be obscured by the accumulation of carbon dioxide has already been mentioned.

Finally, the factor of the intrinsic myocardial integrity should be mentioned. Any pre-existing arrhythmia has been interpreted by Reid[5] as evidence of a relative hypoxia in some part of the heart muscle, and he states that all general anesthetics are potentially dangerous to the muscle itself.

Protective Measures

A review of the reported cases of cardiac arrest shows that they fall into one of three categories. Some have occurred during induction[5], are sudden, and suggest that sympathetic reflex action plus adrenaline and perhaps hypoxia are the precipitating factors. Others have occurred suddenly at the completion of, or soon after completion of the anesthesia and also have suggested reflex action, as when the endotracheal tube is being sucked out or removed, as reported by Shumacker and Hampton[11]. With this suction, they have shown that the oxygen content of the blood drops markedly and suddenly, and it is not unreasonable to suppose that this fact, coupled with the vagal reflexes after the atropine has worn off, can lead to cardiac standstill, or this group may fit the post-hypercapnic phenomenon of Miller and others[12b]. In the third category are cases in which the anesthesia was marked by some difficulty all along. Often the patient became cyanotic, had mucus, was changed from one anesthetic agent to the other, had a sudden drop in blood pressure from blood loss, or began to have an unexplained bradycardia followed by a bigeminal rhythm. In this group, apparently, a multitude of factors were at play before the heart finally ceased. They may be summarized, however, as poor respiratory exchange and poorly maintained blood pressure and blood flow, leading to asphyxia, increased vagal tone, and the cumulative deleterious effects already mentioned.

In view of these factors the following suggestions are made for more positive protection of the integrity of the heart action during anesthesia.

1. It is the duty of the surgeon and his medical colleague to bring the patient to anesthesia in the best condition possible. In the presence of heart disease, this would include digitalization if failure has been present, and perhaps the administration of quinidine in the presence of an arrhythmia. A definite danger in the use of Demerol and intravenous procaine along with quinidine has been demonstrated[13] and should be borne in mind. Electrolyte balance, with particular attention to the potassium and calcium levels should be ascertained, and adequate blood volume and hemoglobin assured.

2. Premedication should include atropine, with the knowledge that its effect may not last more than two hours and may need to be repeated. Furthermore, the anesthetist should be aware of the possibility of hypersensitivity or abnormal depression from the usual doses of narcotics and be prepared to assist respiration from the very beginning of anesthesia, even if it is just a spinal or local.

3. Adequate pulmonary ventilation requires a patent airway and an adequate res-

piratory exchange to remove any excess carbon dioxide as well as to maintain the oxygen supply. Don't mask the accumulation of carbon dioxide by giving too high a concentration of oxygen. Change the soda lime frequently not only to aid the removal of carbon dioxide, but also to decrease the accumulated heat in the system. Check the valves, check the airway, and if there is any doubt, assist the respirations. If adequate respiratory exchange cannot be obtained because of some obstruction, notify the surgeon and change the position of the patient, aspirate him, intubate him, or do anything necessary to overcome this difficulty, even if it means much inconvenience to the surgeon and delay in the operative procedure.

4. Treat immediately any cardiac arrhythmia noted. Be on the lookout for this development, depending not only on the peripheral pulse but on a stethoscope strapped to the chest near the heart so that it can be heard at all times. Treatment may consist of assisting respirations, re-inflating the lung in chest cases, giving intravenous procaine or procaine amide, and, if cyclopropane is being used, changing to ether. If bradycardia occurs in the presence of a good respiratory exchange, consider giving atropine again.

5. Avoid manipulation during light anesthesia, especially in and about the trachea; and prior to tracheal aspirations give high concentrations of oxygen for a few minutes.

6. Insure adequate blood volume replacement, not depending solely on the blood pressure maintenance as evidence of such replacement.

Summary

As much emphasis should be placed on the prevention of cardiac arrest as upon its treatment.

A knowledge of the fundamental physiology of the heart's action and of the factors that influence it during anesthesia is necessary for the prevention of cardiac arrest.

Of all the factors involved, three stand out as basically important: the prevention of hypoxia, the prevention of hypercapnia, and the prevention of severe vagal or sympathetic reflex stimuli to the heart.

A positive program for preventing cardiac arrest is suggested. In brief it is: (1) Keep the oxygen saturation normal with a normal blood volume, (2) keep the carbon dioxide content of the blood down, and (3) control the nervous reflexes with adequate anesthesia and atropine.

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Discussion

Dr. R. E. Spencer (Greensboro): This has been an excellent paper. The points in physiology that have been brought out have been excellent. I think the whole matter boils down to one basic factor, however. Aside from reflex activity—which very seldom, I believe, causes cardiac arrest—the situation hinges on one word, and that is "hypoxia," or "anoxia."

Dr. Leonard Nanzetta (Winston-Salem): Dr. Shaffner has given an excellent presentation of the factors involved in the prevention of cardiac arrest during anesthesia. Certainly a most important factor is the anesthetist. He must have a thorough knowledge of respiratory physiology and a complete understanding of the pharmacology of the drugs he uses. He must possess the technical skill and alertness to apply his knowledge effectively.

Unquestionably, the current practice of using several drugs during a single anesthetic procedure has resulted in cardiac arrest. We had cardiac arrests before the advent of the so-called modern anesthetics, but the accidents were called "death from anesthesia," or "status thymicolymphaticus." Drugs used

in anesthesia, whether they be given orally, intravenously, intrathecally or by inhalation are the tools of the anesthetist. Their value, as well as their dangers, is directly proportional to the knowledge and skill of the person using them. Not so many decades ago the surgeon's armamentarium consisted of a knife, a saw, a few artery clamps, and a tourniquet. Witness the operating room set-up for many of his operations today. There may be literally hundreds of instruments to simplify or even make possible his job. If he injures a common bile duct, or cuts an aorta, is it the fault of the instrument? Likewise, if a patient is given too much or too many drugs, or if after he has received a muscle relaxant his respiratory functions are improperly managed, is it the drug's fault?

Many modern anesthetic drugs mask the clinical signs of physiologic disturbances which may result from their improper use. The patient given barbiturates intravenously or curare does not exhibit hyperpnea with an increased carbon dioxide tension in the inspired atmosphere. Certainly, the qualified anesthetist knows this fact and takes measures to avoid excess carbon dioxide accumulation, which, as Dr. Shaffner has pointed out, is detrimental to the cardiorespiratory mechanism. The most profound effects of carbon dioxide accumulation often do not become evident until the anesthesia is terminated.

As with all accidents, the best treatment for cardiac arrest is prevention. Cardiac arrest rarely, if ever, occurs that hypoxia is not the basic underlying factor. True, an overdose of an anesthetic drug may produce cardiac arrest, but only after the integrity of the cardiac mechanism has been compromised sufficiently to produce hypoxemia, and after sufficient warning has been given the alert anesthetist to take corrective steps. Cardiac arrest does follow certain reflex disturbances, usually vagal in origin. I feel, however, that the cardiac arrhythmias produced by vagal stimulation become relatively benign in the well oxygenated, well ventilated, and properly atropinized patient.

To the factors which contribute to cardiac arrest discussed by Dr. Shaffner I would like to add (1) the "three-minute scrub" and (2) the overloaded operating schedule. Three minutes is scarcely enough time to place a patient in position on the operating table, check his blood pressure, pulse, and respiration, start an intravenous drip, reassure him that all will be well, examine his hospital record for any changes which may have appeared since he was seen the evening before, and, incidentally, to anesthetize him to a safe surgical plane. There are few anesthetists who have not been confronted with the "eager-beaver" resident who wants to get as much of the operation done as possible before the chief comes in, or the very busy surgeon who, rushing to get to another case at another hospital, performs his three minute ablutions as the patient is being wheeled into the operation room, then walks in, champing at the bit to start the operation. Undue pressure is thus often put upon the anesthetist to hurry, and as a result the patient may be rapidly plunged into a dangerous situation. The rapid production of deep anesthesia and profound muscle paralysis—which is easily accomplished with the aid of our intravenous agents—does not allow the adaptive mechanisms of the cardiorespiratory system to compensate proportionately for the sudden onslaught. Severe hypotension ensues. Add to this a possible difficulty with the airway in an apneic patient, then add hypoxia to an inadequate cardiac output, and the stage is set for a case of cardiac arrest which could have been prevented.

It is fortunate for the practice of anesthesia that ether was used first. It was impossible to get the irritating vapor into the patient too fast, and his struggling precluded his being attacked with the scalpel too soon.

DIAGNOSIS AND TREATMENT OF ORAL MONILIASIS

CARL N. PATTERSON, M.D.

DURHAM

Oral moniliasis has become an increasingly frequent clinical diagnosis since the advent and widespread use of the broad spectrum antibiotics. This disease appears to be a direct sequela of antibiotic therapy. The microorganisms in the mouth and throat are in constant competition for survival, and infection occurs when there is an upset in this complex microbiologic balance. Since most of the known antibiotics which kill bacteria are produced by fungi, it becomes apparent why these drugs rapidly upset the normal flora and allow fungi and other nonsusceptible organisms to grow unmolested.

Reports of serious local and general mycotic infections following the use of these antibiotics have become more frequent. In the treatment of brucellosis with aureomycin and chloramphenicol, Harris[1] noted 24 patients with oral and pharyngeal lesions believed to be due to the drugs. Woods, Manning, and Patterson[2] reported 20 cases of oropharyngeal moniliasis following the therapeutic use of penicillin, aureomycin and chloramphenicol. Tomaszewski[3] reported side-effects, including oral moniliasis, in 126 patients treated with aureomycin and chloramphenicol. Kunstadter and his co-workers[4], and Zimmerman[5] each reported a case of mycotic endocarditis following antibiotic therapy; in each case the causative organism was isolated at autopsy and was found to be *Candida albicans*. In April, 1951, the Council on Pharmacy and Chemistry[6] of the American Medical Association requested that a warning label be placed on aeuromycin, Terramycin, and chloramphenicol to inform the physician of the possibility of suppressing the susceptible bacteria and allowing the overgrowth of yeast-like organisms leading to secondary complications.

Read before the First General Session, Medical Society of the State of North Carolina, Pinehurst, May 12, 1953.

From McPherson Hospital, Durham, North Carolina.

Etiology

This recent increase in monilial infections has accelerated investigations into their probable cause. Pappenfort and Schnall[7] reported a growth-stimulating effect on *C. albicans in vitro* by the contents of 250 mg. capsule of aureomycin. Lipnik and his co-workers[8] stated, however, that the effect was "due to an inert carrier in the capsule" rather than to the aureomycin. Woods and his co-workers[2], reporting on controlled *in vitro* studies, failed to find a growth stimulating factor in aureomycin, penicillin, or chloramphenicol.

Table 1
In Vitro Studies of Four Strains of Candida*

	Tubes Containing Antibiotics, Sabouraud's Broth and C. Albicans			*C. Stellatoidae*
	Candida I	*Candida II*	*Candida III*	*(non-pathogen)*
Aureomycin†				
100	0.015	0.01	0.018	0.01
50	0.015	0.01	0.018	0.01
5	0.015	0.01	0.018	0.01
C‡	0.015	0.01	0.018	0.01
Penicillin‡				
5	0.025	0.012	0.028	0.01
2.5	0.025	0.012	0.028	0.01
0.5	0.025	0.012	0.028	0.01
0.25	0.025	0.012	0.028	0.01
C‡	0.025	0.012	0.028	0.01
Chloramphenicol§				
1	0.022	0.011	0.028	0.01
0.5	0.022	0.011	0.028	0.01
0.25	0.022	0.011	0.028	0.01
0.1	0.022	0.011	0.028	0.01
C‡	0.022	0.011	0.028	0.01

*Readings are in terms of milliliters of centrifuged sediment in Hopkins vaccine tubes after 48 hours. Additional controls consisting of antibiotic and broth only produced no sediment.

†Micrograms per cubic centimeter.

‡A control consisting of organism and broth only.

§Units per cubic centimeter.

Smith[9] has suggested that the new infectious syndromes that are occurring in various parts of the body are due to a change in the microbiologic flora of the involved area. He stated that the flora is kept in careful balance by the constant deadly warfare between the gram-positive and gram-negative bacteria, and between these groups and the yeast and mold-like fungi. When the ecologic flora is changed radically, certain organisms multiply rapidly in an area of the body not normally inhabited by that organism or in an area where multiplication has been inhibited previously by biologic antagonists. This overgrowth of organisms may produce disease without specifically lowering the patient's resistance.

Diagnosis and Treatment

C. albicans is an inhabitant of mucous membrane-lined cavities and tracts in many normal individuals, and the simple isolation of the organism does not warrant the diagnosis of moniliasis. However, in observing many cases of oral disease, we have noted 100 cases which by history seemed due to various antibiotics. In these cases, *C. albicans* was constantly found to be the predominating organism. This disease presents a consistent clinical picture regardless of the mode of administration of the offending drug. The picture varies according to the stage in which the case is observed. The tongue is most frequently involved. At first it is thickly coated with an overgrowth of the filiform papillae, giving the appearance of a brown or black hairy tongue. Later the coating or hairy covering comes off, leaving the tongue dry, swollen, and beefy red, with the fungi-form papillae standing out in re-

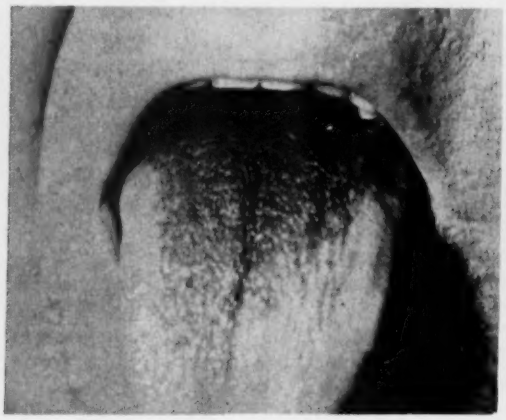

Fig. 1. Typical picture of a hairy tongue caused by Monilia albicans.

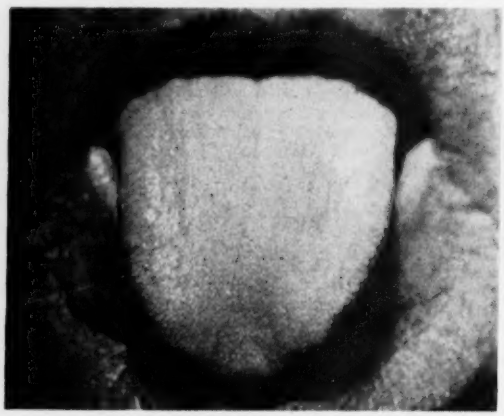

Fig. 2. Photograph of tongue after treatment with 2 per cent Asterol Citrate.

lief. The tongue is tender to palpation and to contact with acid juices and foods. The sense of taste is markedly altered. The buccal mucous membrane is at first covered with circumscribed or coalesced areas of whitish exudate which can be wiped away easily. In 24 to 72 hours this milk curd-like exudate comes off and the buccal mucous membrane is left dry, glazed and parched. There are often small petechial hemorrhages in the mucous membrane. Similar exudate has been seen less frequently on the tongue, soft palate, uvula, arytenoids, aryepiglottic folds, and pyriform fossae. This is followed later by a dry, glazed, reddened edematous stage. In one instance, marked esophagitis with stricture was noted.

All lesions observed followed antibiotic therapy, and clinically the findings have been so consistent as to suggest a disease entity which occurs as a sequela of antibiotic therapy. Whether this is purely a fungus disease or an alteration of the microbiologic flora with *C. albicans* as the predominating organism will have to be investigated by careful bacteriologic studies. It has been found, however, that either *Monilia albicans* in association with gram-negative bacteria has been the predominating organism, or a pure culture of the Candida has been obtained. Whichever the case may be, elimination of the subjective symptoms and clearing of the objective findings are dependent upon the suppression of the fungi. Fortunately, the majority of the cases that do occur subside promptly when the offending drug is discontinued. However, those that do persist have been most resistant to treatment. Vitamins, gentian violet, and 1-200 formaldehyde solution produced only fair results. It was noted, however, that the subjective and objective findings were directly proportional to the fungi present in the mouth and throat.

Stritzler and associates[10] found that monilial infections of the glabrous skin and nails responded more favorably to Asterol Dihydrochloride than to any other medication, and it was decided to try this preparation topically in the treatment of oral moniliasis.

The citrate of Asterol was substituted for the dihydrochloride, as the former lent itself more readily to preparing a pleasant-tasting aqueous solution. Prior to clinical trial, Asterol Citrate* was investigated extensively in the Pharmacology Department of the manufacturer. The findings in animals[11] are summarized briefly as follows:

The LD50 of Asterol Citrate in mice was found to be 750 mg. per kilogram by the subcutaneous and 802 mg. per kilogram by the oral route. These figures compare favorably with the respective LD50 values of 375 mg. per kilogram and 500 mg. per kilogram reported for Asterol Dihydrochloride[12].

No significant difference occurred in the average weight or the physical state of weanling rats given 2 drops of a 2 per cent Asterol Citrate Solution daily for 30 days, when compared to a control group which did not receive the drug. The daily intragastric administration of 20, 40, and 80 mg. per kilogram of Asterol Citrate over a period of one month had little direct adverse effect upon weight maintenance of adult rabbits.

To determine the effect of repeated topical application to the buccal, lingual and oropharyngeal mucosae, these tissues of guinea pigs were swabbed daily with Asterol Citrate solution in concentrations of 2, 5, and 10 per cent five times a week for a 13-week period. On microscopic examination there was no evidence of local tissue damage. Microscopically, however, a slight local inflammatory reaction in the submucosa of the tongue and cheeks was evident, particularly in the animals treated with the higher concentrations; but the epithelium did not appear to be affected. The trachea, bronchi, and lungs, as well as the esophagus and the gastrointestinal tract, showed no injurious effect from swallowed or aspirated drug. Suggestive evidence of gastric irritation was not confirmed by histologic studies. There was some suggestion of renal excretion of the absorbed portion of the drug. The growth response, hemoglobin values, and red cell, white cell, and differential counts in these animals had remained within normal limits.

Instillation of 0.6 cc. of a 2 per cent solution of Asterol Citrate into the conjunctiva of rabbits, produced no noticeable irritation. Similarly, 0.1 cc. of this solution administered intracutaneously on the dorsal side of the ear of this species left no marks of irritation. When 2 cc. were injected into the subcutaneous tissue of the right flank of the rabbit no signs of inflammation, infiltration, or other symptoms of irritation were seen.

*The 2% Asterol Citrate Solution (aqueous) used in this study was furnished by Hoffman La-Roche, Inc., Nutley, New Jersey.

Use of Asterol Citrate in 31 Cases

In view of the pharmacologic data indicating that a 2 per cent solution of Asterol Citrate should provide a non-irritating topical medication of low toxicity, an investigation of the clinical effectiveness in oral monilial infections was undertaken in a series of 50 patients.

In 31 cases sufficient clinical and cultural follow-up was obtained to give a report on the effectiveness of this drug. All cases met the following suggested criteria for the diagnosis of oral moniliasis:

1. The typical clinical picture of stomatitis which produces subjective symptoms
2. Isolation of *C. albicans* as the predominating organism grown on Sabouraud's broth and corn meal agar.

All cases studied followed the use of antibiotics either by local application (particularly in the form of lozenges) or systemic administration, and all patients were adults. The duration of symptoms in this group varied from 1 to 16 weeks, with an average duration of 3 weeks.

The method of treatment, after the diagnosis had been established, was to swab the involved areas with cotton applicators moistened with 2 per cent Asterol Citrate. If the tongue was involved, as was often the case, it was scrubbed vigorously with the drug. Finally the patient rinsed his mouth with about ½ oz. of the solution. At least three minutes were taken for each treatment. This procedure was repeated every other day in the out-patient clinic, but was used twice a day in hospital patients. In most of the cases the 2 per cent solution was diluted with sterile water 1:3 parts. The patients were instructed to use it at home three times a day and were cautioned not to swallow the drug.

In no case was any evidence of local irritation or general toxic reaction noted. Most patients liked the taste and none found it objectionable. Subjectively, relief of symptoms was often obtained after the first or second treatment.

We have had no toxic manifestations in our adult group; however, several investigators[13] have reported encephalopathy in infants and young children during therapy with Asterol Dihydrochloride. Since the drug seems to be potentially neurotoxic, the manufacturer does not recommend its use in children under 3 years of age, as it might inadvertently be swallowed and cause systemic reaction or be absorbed from the mucous membranes.

Results

Table 2 shows the results obtained in 31 cases of oral moniliasis. Twenty-nine cases (93.6 per cent) were rendered asymptomatic; 2 cases (6.4 per cent) were improved. Both of the latter patients continued to complain of a sore, burning mouth and tongue; however, no objective evidence of disease could be found. Vitamin therapy resulted in no further improvement.

Table 2
Results Obtained with 2% Asterol in 31 Cases of Stomatitis Following Antibiotic Therapy

Case No.	Area of Involvement	Duration of Symptoms	Duration of Treatment (days)	Results
1.	Tongue and buccal mucous membranes	4 months	5	Improved
2.	Tongue, buccal and pharyngeal mucous membranes	3 weeks	3	Asymptomatic
3.	Tongue, palate, uvula	10 days	5	Asymptomatic
4.	Tongue, buccal and pharyngeal mucous membranes	5 weeks	4	Asymptomatic
5.	Tongue and pharyngeal mucous membranes	2 weeks	3	Asymptomatic
*6.	Tongue, buccal and pharyngeal mucous membranes	2 months	8	Asymptomatic
7.	Tongue and buccal mucous membranes	10 days	1	Asymptomatic
8.	Buccal, pharyngeal and laryngeal mucous membranes	1 week	4	Asymptomatic
9.	Tongue and pharyngeal mucous membranes	2 weeks	1	Asymptomatic
10.	Tongue, buccal and oral mucous membranes	2 weeks	3	Asymptomatic
11.	Tongue, buccal, palatal and pharyngeal mucous membranes	1 week	2	Asymptomatic
12.	Tongue and oral mucous membranes	4 weeks	5	Asymptomatic
13.	Tongue, soft palate, and epiglottis	2 weeks	11	Asymptomatic
14.	Buccal, oral and pharyngeal mucous membranes	10 days	5	Asymptomatic
15.	Tongue, buccal mucous membrane and soft palate	2 weeks	11	Asymptomatic
16.	Tongue and oral mucous membranes	2 weeks	10	Improved
17.	Tongue and soft palate	10 days	4	Asymptomatic
18.	Tongue and soft palate	2 weeks	7	Asymptomatic
19.	Tongue and soft palate	3 weeks	7	Asymptomatic
20.	Tongue	1 week	1	Asymptomatic
21.	Tongue	1 week	1	Asymptomatic
22.	Tongue and oral mucous membranes	4 weeks	5	Asymptomatic
23.	Tongue and buccal mucous membranes	6 weeks	3	Asymptomatic
24.	Tongue, oral, pharyngeal and laryngeal mucous membranes	8 weeks 4 weeks	6	Asymptomatic
25.	Tongue and epiglottis		3	Asymptomatic
26.	Tongue, oral, perleche, pharyngeal, nasal, vaginal and perianal membranes	6 weeks	12	Asymptomatic
27.	Tongue, oral and buccal mucous membrane	10 days	7	Asymptomatic
28.	Tongue, oral and pharyngeal mucous membranes	2 weeks	12	Asymptomatic
29.	Soft palate and pharyngeal mucous membranes	1 week	7	Asymptomatic
30.	Tongue	10 days	9	Asymptomatic
31.	Tongue, hard and soft palates	4 days	10	Asymptomatic

*Treated with formaldehyde without relief

Prior to the studies on Asterol Citrate, an aqueous solution of 1:200 formaldehyde solution was used in the treatment of oral moniliasis. Fifteen cases which fulfilled the criteria for diagnosis were treated and follow-

Fig. 3. (Case 2). Oral moniliasis with associated skin lesions. Note particularly the crusting around the mouth and the dry glazed tongue.

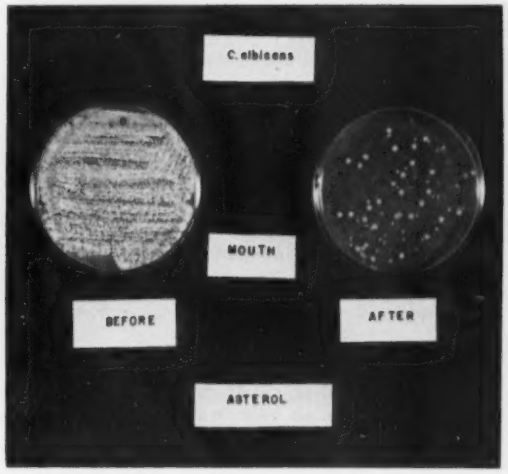

Fig. 4. Cultures of Monilia albicans taken before and after treatment with 2 per cent Asterol Citrate.

up studies obtained. The technique was the same except that all patients were instructed to use this solution at home three times a day. A comparison of the results of the two drugs is shown in table 3. (Note the average number of days necessary to render the patient asymptomatic.)

Table 3

Cases	Asymptomatic	Days	Improved	Days	Unimproved	Days
Asterol Citrate—						
31*	93.6% (29)	5.3	6.4% (2)	7.5	0	
1:200 Formaldehyde Solution—						
15	53 % (8)	8.5	20 % (3)	16.0	27% (4)	15.5

*Cases 1 and 6 in table 2 were treated with 1:200 formaldehyde solution without relief.

Report of Cases†

Case 1

An 18 year old white youth was admitted to McPherson Hospital with the complaint of sore throat of 10 days' duration. He had been given one intramuscular injection of procaine penicillin and started on aureomycin, 250 mg. every six hours for 16 doses, by his local medical doctor. His throat improved for 24 hours, but in 48 hours it became much worse.

On admission he was extremely toxic, with a temperature 101 F. Physical examination was negative except for his mouth and throat, which showed marked hyperemia of the oral, buccal, and pharyngeal mucous membranes. On the soft palate and pharyngeal wall were several irregular lesions covered with a milk curd-like exudate showing a tendency to coalesce. Smears and cultures were taken for predominating organisms, diphtheria, and fungi. The immediate smear was loaded with yeast and showed occasional gram-negative rods. Diphtheria and infectious mononucleosis were considered because of the extensive palatal and pharyngeal involvement. Crystalline penicillin 100,000 units every three hours was given for 18 hours and then discontinued, as the Loeffler's slant showed no evidence of diphtheria. The oral lesions were treated with topical applications of 2 per cent Asterol Citrate twice a day. Cultures on Sabouraud's slant showed a marked decrease in the number of colonies of Monilia in 24 hours. After 36 hours the patient was able to swallow without difficulty; and after 5 days the mucous membranes were clear except for slight hyperemia, and cultures were negative for Monilia.

Case 2

A 51 year old white woman was admitted to McPherson Hospital on September 16, 1951, with the complaint of sore mouth and throat. Past history revealed that on July 27 the patient was hospitalized for acute appendicitis. Following the operation she received intramuscular injections of penicillin prophylactically. A sore mouth and throat on July 29 developed. On July 31 she began to have a heavy vaginal discharge and perianal rash. Various medications were tried for the vaginal condition, without improvement. Similarly, several different lozenges and applications of gentian violet were used for her throat without relief. Her condition grew progressively worse.

Admission examination revealed a dehydrated 51 year old white woman. Around the lips was a maculo-papular rash with a marked perleche. Some of the lesions were crusted. The lips bled on motion. The tongue was devoid of filiform papillae—dry, glazed, swelled and exquisitely tender to palpation. The buccal mucous membranes were covered with milk-curd exudate. The pharyngeal and hypopharyngeal walls were dry and glazed. A dermatologic consultation revealed vaginal, perineal, and perianal inflammation. Cultures from buccal and vaginal mucous membranes showed a pure culture of *Candida albicans*. A routine specimen of urine showed Candida, but analysis of a catheterized specimen did not confirm the finding.

The patient had no appetite; food had no taste, and it was painful to chew even semi-soft foods. The oral lesions were painted twice a day with a 2 per cent solution of Asterol Citrate. In 48 hours the oral lesions were markedly improved. The patient

†Cases 1 and 2 are referred to in table 2 as cases 14 and 26.

could chew semi-solid foods without much pain; however, the sense of taste was still impaired. On the sixth hospital day the patient complained of discomfort in the left naris. Examination revealed a crusted area in Kiesselbach's triangle. Removal of the crust disclosed a red area, about 1½ cm. in diameter, which extended into the skin of the naris. A culture taken from this area revealed *C. albicans*. The area was treated with Asterol Citrate.

The patient was discharged on her twelfth hospital day. On this date cultures from the mouth and nose were negative for *C. albicans*. On examination three weeks after discharge, the oral mucous membranes were normal in color. The tongue appeared normal, the sense of taste had returned to normal. There was still slight fissuring at the corners of the mouth. Three weeks later the patient was completely asymptomatic.

These cases represent two extremes. Case 1 represents an acute fulminating stomatitis with evidence of marked toxicity which necessitated a differential diagnosis. Case 2 represents chronic stomatitis with associated extensive vaginal and skin lesions. Each of these patients responded completely to the topical application of 2 per cent Asterol Citrate.

Comment

From clinical observation and cultural studies, oral moniliasis seems to be a disease of increasing frequency. The majority of cases subside after the offending drug is discontinued. These cases need no specific therapy. If, however, symptoms have not improved after five to seven days, specific therapy in the form of an antifungal drug seems indicated.

The results observed with Asterol Citrate have been dramatic in some cases. Often one application of the drug has brought complete relief of the subjective symptoms. Cultures taken before therapy is instituted and then repeated in 24 hours show marked decrease in the number of fungus colonies present. The subjective symptoms seem to vary in direct proportion to the number of fungus organisms present in the mouth. One would suppose the antifungal drug temporarly depresses the fungi and allows the normal bacterial antagonists to grow unmolested, and thus the microbioligic flora of that individual is re-established.

Conclusion

1. Monilial stomatitis is a definite disease entity which has increased in frequency since the use of wide spectrum antibiotics.

2. Certain criteria are suggested for the diagnosis of this disease.

3. A new drug, 2 per cent Asterol Citrate, has been used in the treatment of 31 cases of oral moniliasis. This series is not sufficient to evaluate the effectiveness of Asterol Citrate in monilial stomatitis conclusively; however, it has been the most efficient antifungal agent I have yet used for the treatment of this disease.

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Vitamins

The amount of energy, labour, material, and advertising designed to persuade the public to consume, and the medical profession to prescribe, a daily dose of vitamins of various kinds is phenomenal. The argument runs somewhat like this: "Pain in the little finger is an early sign of vitamin-blank deficiency. Therefore, anybody who ever gets a pain in the little finger should take vitamin blank daily. Even if you haven't got a pain in your little finger vitamin blank taken daily will prevent you getting one." It is also unfortunate that the word vitamin should suggest that it is somehow connected with giving vitality.

An official statement from an authoritative source on the limitations of vitamins would not come amiss. In its absence I will stick my neck out with the following pronouncement. A daily supplement of vitamins is valueless to the normal adult. In particular there is no evidence to show that it will prevent coughs and colds (American papers, please copy). The high-dosage vitamin treatment of certain diseases where the vitamin is used like a drug is of course an entirely different subject.—Lewis, A.: Letter to the British Medical Journal, Page 729, March 28, 1953.

DIAGNOSIS IN CARCINOMA OF THE ORAL CAVITY

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The problem of cancer of the oral cavity has not been solved, and it is becoming more complex each year. It accounts for about 6,000 deaths annually, and together with cancer of the lip, comprises 6 per cent of all types of cancer, being mostly of the squamous cell variety. The paradox of this disease is that, while next to skin cancer it is the tumor that may be most easily diagnosed in its early stage, 75 to 80 per cent of the patients come to definitive treatment after it has spread beyond the confines of the local lesion. We must discover the causes of this lethal paradox and eliminate the diagnostic tardiness.

Analysis shows that early diagnosis of mouth cancer rests upon three pillars: first and most important, recognition by the patient of the symptoms that cause him to seek medical aid; second, education of the public as to the nature and significance of various symptoms; third, recognition by the physician or the dentist who *first* sees the patient. It must be remembered that about 30 per cent of all patients with mouth cancer consult a dentist first[1]. Delay in diagnosis may result from a fault in any one of the three pillars, with a serious loss of time during which the tumor may advance.

Physicians as a group may support public health programs designed to promote case-finding and subscribe financially to the American Cancer Society and endorse its good works; but they can make their greatest contribution by eliciting an accurate history and performing a complete physical examination whenever a patient presents himself for treatment. The oral cavity has received a minimum of attention by the medical profession—usually only a cursory physical examination, with little attempt to question the patient about his oral symptoms. Consequently, many easily visualized cancers of the oral cavity escape notice, only to emerge later as advanced tumors.

Further, there is little indication that medical students receive sufficient instruction in examination of the oral cavity. Several recent textbooks on medicine, surgery, and physical diagnosis contain little that is helpful in orienting the student and teaching him the rudiments of examination. Moreover, not only is it difficult for the novice to examine the oral cavity adequately, but the methods of choice and purposes of the examination are rarely recorded in print for reference.

For the past two years, in the Medical School of the University of Pennsylvania, an extensive course entitled "Examination of the Patient" has been instituted for second year students. Every aspect of physical examination is presented by clinicians in the various fields, so that, in addition to the usual instruction by internists, the methods used by neurologists, gynecologists, surgeons and other specialists are offered to the students. The students also perform examinations on hospital patients during the same period. By this means they become familiar with the details of the "routine" complete physical examination, applied with proportionate emphasis to all parts of the body. The examination of the oral cavity is included in this procedure, and to it all students devote a total of three hours in demonstrations and clinical work, out of a total of 108 hours for the entire year course.

Unfortunately, we possess no new ways of diagnosing oral cancer. The smear technique has never been applied to this area, and there exists no means of screening patients. We must confine ourselves to the history and the use of the senses, aided by a few simple instruments and by a high index of suspicion. When the tumor has been located, biopsy must be performed to confirm the clinical diagnosis.

Signs and Symptoms

The early signs and symptoms of oral cavity cancer are minimal and usually cause little or no discomfort. The presence of a tumor, ulcer, fissure, or crust may be noted in the *visuable areas. Vermilion border* lesions are seen earliest. In the *posterior regions*, visibility is reduced or absent, and sensation is not as acutely perceived as it is anteriorly. For that reason, the earliest symptoms often go unrecognized. The patients have difficulty in localizing the lesions even though they are aware of "something there." Lesions of the nasopharynx

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characteristically cause unilateral deafness, tinnitus aurium, or stuffiness in the back of the nose on one side. In the nasopharynx, at the base of the tongue and lateral pharyngeal walls, "sticking," burning or dysphagia is produced by neoplasms. Hoarseness, cough and aphonia are symptoms of laryngeal lesions.

Pain is not an early symptom of oral cavity cancer but it may be the first symptom occasionally in "silent" tumors which arise and spread without the knowledge of the patient. This is particularly true of lesions of the hypopharynx. There are certain other "silent" tumors, especially the lymphoepitheliomas of the nasopharynx, which often exhibit cervical node metastasis as their first sign or symptom. In our present state of knowledge we are unable to detect these lesions in an earlier phase of their existence.

Physical Examination

A standard method of examing the oral cavity should be followed, much as in the case of the chest or cranial nerves. In patients who have oral symptoms, the regular examination should be supplemented by more thorough methods. Inspection and palpation are the two means employed, and later the use of special instruments is required. Reflected light from a head mirror provides the best source of illumination, and allows free use of both hands.

First, inspect the vermilion borders, and then examine all of the visible oral mucosa. Special care must be taken to visualize the hard palate, buccal sulci, floor of the mouth, and base of the tongue. The state of the teeth should also be recorded. In using the tongue depressor to expose the surfaces, care must be taken not to allow it to cover a small tumor. Next, and perhaps most important, all the above mentioned areas are palpated. The finger is placed inside the mouth and the thumb used externally in coordination to examine the cheek tissues. The base of the tongue, tonsils, soft palate, lateral pharyngeal walls, and epiglottis are easily palpated. Early carcinomas are sometimes hidden in invisible submucosal tissue discoverable only by palpation.

Bimanual palpation is employed to advantage in examining the structures of the floor of the mouth and parotid gland. The *right* index finger is inserted along the *right* floor of the mouth, and the left hand palpates the right submaxillary structures. The submaxmillary salivary gland is then completely palpated, and any enlarged lymph nodes and other masses are easily examined. The hands should be reversed when shifting to the opposite side or else the position becomes awkward and the examination inaccurate.

More complete examination is reserved for patients who have one or more of the symptoms enumerated above. It consists of the use of instruments which are familiar to everyone and which can be purchased at low cost and kept available in the office. The head mirror has already been mentioned. Laryngeal mirrors, large and small, tonsil pillar retractor and metal tongue depressor can be used expertly with practice. By this means the recesses of the pharynx, as well as the larynx and choana may be visualized. For a more accurate view of the nose, the nasal speculum and nasopharyngoscope must be obtained. The latter is expensive and is not necessary except for those particularly interested in the field.

Some patients are ideal for examination, while others gag excessively. In the latter, the topical application of 2 per cent pontocaine spray is effective in reducing reflexes. When the pharynx is fully anesthetized, the muscles are relaxed and palpation greatly enhanced.

Extent of Lesion

In order to determine the type and details of treatment, the extent of each cancer must be outlined. Therefore, there remains a secondary problem in diagnosis after finding the lesion and performing biopsy. Lateral and deep spread, and origin from multiple centers must be determined before therapy can be definitely planned. Occasionally it becomes necessary to administer a general anesthetic to obtain this information.

The use of roentgen techniques is mentioned here as a valuable supportive aid in diagnosing oral cavity carcinoma, especially in the pharynx and larynx. It is beyond the scope of this paper to enlarge upon the radiologic aspects.

Conclusion

I make no apology for presenting a plan of diagnostic procedure in a region where diagnosis should be made so easily. It is the function of organized medical groups to advance public knowledge of the symptoms of

disease, and to act as case finding agencies. The physician must discharge his duty by performing a thorough examination and making an accurate diagnosis.

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THE USE OF CHEMICALLY INDUCED HYPOTENSION IN THE TREATMENT OF INTRACRANIAL ANEURYSM

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In a continuing study of the general problem of intracranial bleeding conducted at Duke Hospital during the past five years, the pathogenesis, the varying clinical syndromes, and the intravascular cerebral pressure relationships as noted in over 300 patients have been discussed[1]. The present report has to do with studies conducted on 26 patients with intracranial aneurysms or large meningiomas, in whom hypotension was induced by chemical means for the purpose of controlling hemorrhage during the operative conduct of such difficult lesions.*

Materials and Method

Because of the admonition that the technique should be employed only in the presence of specific and urgent indications, and not for mere surgical convenience, the selection of patients was dictated by the knowledge that severe operative bleeding might be anticipated during the surgical intervention[2]. Ten patients were proved to harbor congenital cerebral aneurysms or arteriovenous anomalies; 7 patients had massive meningiomas, and the remainder gliomas in whom lobe resection appeared a probable means of treatment. Pendiomide (pentamethyl-diethyl-3-aza-pentane-1, 5-diammonium-dibromide) was used in 16 cases, C_6, an hexamethonium chloride compound, was used in 8 cases; and Arfonad, a trimethylene thiophanium compound, was used in 2 cases.

The efficacy of induced chemical hypotension in the control of operative hemorrhage is a proved fact[3]. Since cerebral complications have been reported as one of the major hazards of this technique[2], it seemed desirable in this study to obtain data, when possible, concerning intravascular pressure in cortical vessels, as these pressures might be related to arm cuff and systemic pressures. Accordingly, all patients were studied in the following manner. In the preoperative period, liver and kidney function studies were obtained and electrocardiograms were recorded. After surgical anesthesia was induced, the radial artery was cannulated and direct arterial pressure recorded continuously on a Sanborn recording electromanometer. These readings were compared to arm cuff pressures taken every three to five minutes. Continous electrocardiograms were obtained by means of a Sanborn Visocardiette. When the brain was exposed, a suitable cortical artery was cannulated with a no. 27 needle, and continuous pressure readings were recorded either at intervals or continuously. Chemical hypotension was induced with the trunk and head elevated 30 degrees before the scalp incision was commenced. In earlier cases, for study purposes, hypotension was not induced until the cortex had been exposed. The effect of the normotensive and hypotensive states were noted in the cortical and peripheral arterial pressures and a clinical evaluation made of the degree of hemorrhage and the ease with which it could be controlled.

Two case reports will suffice to illustrate particularly this second point.

Illustrative Cases

Case 1 (Duke Unit No. D-58287)

Although this patient, a 39 year old white man, had noted infrequent bifrontal headaches for all of his adult life, he had been quite well until five weeks prior to admission when he developed a severe agonizing pain above and behind the right eye which shortly developed into a generalized right-sided headache. This headache lasted four to five hours and then gradually subsided, without sequelae. The patient was thought to be well again until three and one-half weeks prior to admission when he experienced similar pain, which continued to throb constantly

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until admission. About 30 minutes following onset of his second bout of head pain, he noticed that he was unable to open or to move the right eye. When he lifted the right eyelid, diplopia was noticed upon right lateral gaze. The head pain had been persistent, and had been associated with marked nausea and vomiting for the first week after its onset, and again during the past five days before admission. Mild hypertension had been noticed by his family physician for the past 12 months.

General physical examination showed a well nourished, quite alert white man of 39. His blood pressure was 170 systolic and 100 diastolic, and there were no abnormal changes in the general physical review.

The neurologic examination showed a supple cervical spine. Vision was reduced in the right eye to 20/40, while vision in the left eye was normal. There was complete paralysis of the third and fourth nerves on the right side, with ptosis, a dilated fixed pupil, and inability to move the orbit except in a lateral plane. Lumbar puncture showed a subarachnoid pressure of 160 mm. of water, and there was no evidence of subarachnoid hemorrhage.

A right internal carotid arteriogram revealed an aneurysm at the junction of the posterior communicating and internal carotid arteries of the brain. Under Pendiomide induced hypotension (fig. 1), the major neck vessels were dissected on the right side. The intracranial vascular pressure studies demonstrated, after temporary occlusion of the internal carotid artery, that an adequate cross circulation existed in the Circle of Willis, with only a 30 per cent pressure fall following temporary occlusion. A right frontal craniotomy was then carried out and the aneurysm visualized at the junction of the posterior communicating and internal carotid arteries. The brain was relaxed and exposure was adequate.

At this time, under induced hypotension, the mean radial and cortical intravascular pressures were respectively 64 and 60 mm. of mercury. The aneurysm was freely mobilized from its attachment to the dura. There was but slight oozing from the bulbous portion of the aneurysm, quite different from the usual forceful hemorrhage associated with rupture of such a lesion. It was technically possible to clip the neck of this aneu-

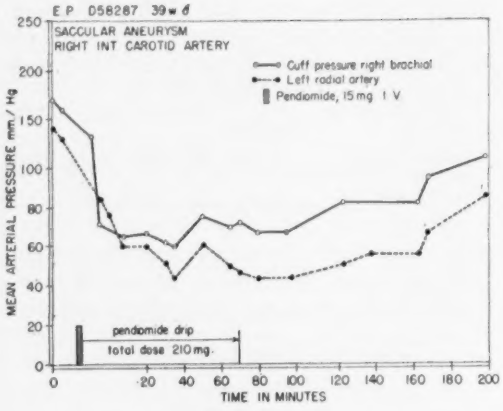

Figure 1.

rysm without interrupting the parent vessels.

The postoperative course was uneventful. A postoperative arteriogram showed a normal vascular system with the clip at the location of the previously demonstrated aneurysm.

Case 2 (Duke Unit No. C-97983)

For a number of years, this white, 20 year old man had noted bifrontal headache, and it was very difficult to delimit the beginning of his present illness. It seemed rather clear that for at least two years this headache had been moderately incapacitating and intermittently severe, and that during these episodes he had sought professional assistance for relief. About 10 months prior to admission, during one of these intermittent episodes of severe bifrontal headache, nausea and vomiting had appeared for the first time and lasted one and one-half days. At the end of this period the patient had had a 20-minute episode of right-sided loss of sensation, beginning in the right foot and progressing proximally to involve the right arm, right side of the trunk, and right side of the face. The most recent episode, quite identical to the present, occurred three weeks before admission. Following this, his thinking had been less clear, he had had some difficulty in speaking, he had been quite lethargic, and he had noted some dimness in vision.

General physical examination in this patient was quite unremarkable. He was rather lethargic but cooperative. He was slow in response to command, and was easily confused. There was no clearcut aphasia, and he seemed to be fairly well oriented.

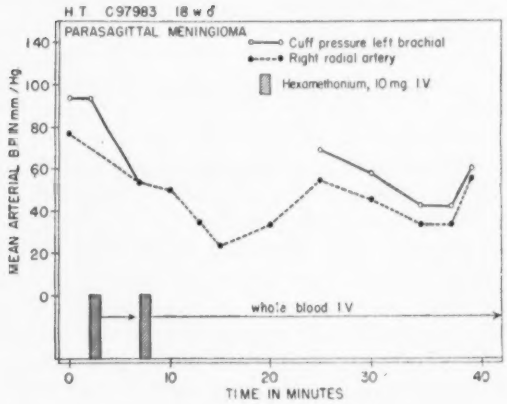

Figure 2.

Neurologic examination revealed bilateral papilledema, with full visual fields except for a suggestion of a lower temporal quadrant defect in the right eye. There was rather marked impairment of cortical sensibility in the right upper extremity, but no motor weakness could be demonstrated. All deep tendon reflexes were hyperactive but equal, and there was a positive Babinski response on the right side.

Among his laboratory studies, stereoscopic lateral roentgenograms of the skull showed extensive changes in the left parieto-occipital area, with irregularity of the inner table and areas of thinning which suggested tumor erosion. There was a suggestion of convolutional atrophy throughout the skull, and the sella turcica was quite large, with the posterior clinoids flattened and apparently eroded. An electroencephalogram revealed abnormal waves present over the entire left occipito-parietal area. Visual fields revealed a right lower homonymous quadrantanopsia, with fairly normal direct vision.

A left carotid arteriogram showed a marked loss of vascular structures in the left parietal area with the middle cerebral artery displaced downward and forward, and an aberrant artery passing caudally sweeping around an apparent mass in the left parietal lobe. Under induced hypotension, with hexamethonium, (fig. 2), a rather massive left temporo-parietal meningioma weighing 385 Gm. was resected. At the depth of the induced hypotension, the intravascular pressure in the radial artery measured 24 mm. of mercury. Tumor resection was completed in 20 minutes, during which 1000 cc. of whole blood were administered intravenously, although there was no obvious evidence of severe blood loss.

Table 1
Average Fall (Per Cent) in Mean Arterial Pressure with Controlled Hypotension

Compound	*Number Cases (Total)*	*Cuff (Per Cent)*	*Radial (Per Cent)*	*Cortical (Per Cent)*
Pendiomide	16	35.4	50.2	41.9
Hexamethonium	8	30.5	61.5	60.0
Arfonad	2	32.7	45.2	52.7
All Compounds	26	34.6	52.9	47.6

His convalescence was quite uneventful, and, upon a later admission, cranioplasty was carried out with a polyethylene plate.

The relationship among cuff pressures, intravascular radial artery pressures, and intravascular cerebral cortical pressures is noted in table 1. These data show the rather significant variation in pressure obtained by the arm cuff method by the attending anesthetist and the actual systemic intravascular arterial pressure as recorded in the radial artery of the same or opposite arm. It is obvious that the actual fall in systemic arterial pressure may be much more profound than that demonstrated by the usual arm cuff method, and may indeed approach a level incompatible with cortical function These observations are particularly pertinent, since it is a common finding that when systemic arterial pressures do reach a critical level, the arm cuff method may fail to portray that pressure reading. With recording of systemic arterial pressures from the radial artery the actual level of induced hypotension is always apparent, and if dangerous levels are reached, may be controlled by reposturing the patient[2c]. If the degree of induced hypotension approaches a critical area, it may be reversed rapidly by the use of such a vasodepressor as Levophed. These studies also indicate the fact that for these purposes the intravascular pressure within the cortex approximates that found within the radial artery.

Comment

These studies of the response of the systemic and cortical pressures to induced hypotension show conclusively that ordinary arm cuff pressures are inadequate to delimit the exact extent or depth of the fall in pressure found in many individual patients. The recording of intravascular radial systemic pressures does offer an adequate means of following pressure changes in terms of depth of induced hypotension and

in terms of the rather rapid changes that may ensue. Furthermore, such radial systemic pressures are more or less identical with pressures produced in cortical vessels. When a satisfactory chemically induced hypotension is obtained, the resulting control of bleeding represents a distinct advantage and aid to the technical requirements that are necessary for the control of critical brain lesions, be they aneurysms or tumors. Unfortunately, the response of all individuals to these ganglionic blocking drugs is quite unpredictable, and an adequate degree of chemically induced hypotension is not obtained with regularity. This unpredictability of response may be a factor which measures the individual patient's response to stress through the production of circulating adrenaline, and is at least a fertile field for further investigation. The concept of chemically induced hypotension for the control of operative hemorrhage, however, is very good, and may be considered an advance in the neurosurgical care of selected patients.

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Atrophic arthritis rarely begins in old age. Most of the patients suffer only from the mechanical faults and muscle atrophy that were incurred earlier in life. Mild exacerbations of the disease in the joints may follow infections of the respiratory tract or a state of malnutrition. Cortisone and ACTH should not be employed because in these old people it causes high blood pressure and psychotic states. Hypertrophic arthritis can be found by physical examination or by x-ray study in all old people. It seems to be productive of no real difficulty except in the hip joint (malum coxae senilis). Myalgias are common from many causes — bursitis, fibrositis, sprains, awkward use and physical unfitness.— Monroe, R. T.: The Effect of Aging of Population on General Health Problems, New England J. Med. 249:323 (Aug. 20) 1953.

PHLEBOTHROMBOSIS

Incidence and Treatment

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The problem of phlebothrombosis and secondary pulmonary embolism is constantly with the physician, whether he be internist or surgeon. The literature is crowded with various reports concerning the incidence, diagnosis, and treatment of these conditions. The mechanics of coagulation of the blood and certain known factors which predispose to thrombosis are generally agreed upon, but the multiplicity of available prophylactic and therapeutic measures somewhat confuse the picture.

Familiarity with the known mechanisms of normal blood coagulation and the various factors that tend to accelerate or retard it is essential to the diagnosis, prevention, and treatment of phlebothrombosis. It is also necessary to know what steps may be taken clinically to prevent or to treat this unwanted complication.

The Mechanics of Coagulation

The fluidity of the blood is maintained by a dynamic equilibrium between two opposing groups of factors within and in contact with it. The anticoagulant factors can be listed as rapid circulation, intact vascular endothelium, antithromboplastin, antithrombin, and fibrolysin. Coagulant factors are slow or stagnant circulation, damaged vascular endothelium, thromboplastin, prothrombin, AC-globulin and fibrinogen[1].

Thrombin, acting as a protective mechanism against hemorrhage, is liberated into the vascular system of patients on whom surgery has been performed or on those who have undergone some trauma. We can assume, therefore, that phlebothrombosis does not occur unless thrombin is present in the vascular system to a greater or lesser degree. However, phlebothrombosis does not develop in all such patients, indicating the presence of a circulating antithrombin which maintains the proper balance in the clotting mechanism.

Ochsner and others[2] found in a series of 201 cases that surgical patients with sustained antithrombin levels of 1:32 or higher

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remained free of intravascular clotting. In patients given alpha-tocopherol, with antithrombin levels of 1:16 or lower and prothrombin times less than 20 seconds, the incidence of phlebothrombosis and fatal pulmonary embolism was the same as in the control series. Their investigations had led Ochsner and colleagues to believe that alpha-tocopherol, which is normally present in alpha and gamma globulin fractions of blood, was a circulating antithrombin factor. Alpha-tocopherol and calcium were given to a group of 43 patients all with antithrombin levels of 1:16 or less, with the result that there were no recognized cases of thrombosis. The investigators felt that the maintenance of a high level of antithrombin will prevent the formation of a new thrombus, but will not prevent the detachment of a previously formed clot.

Weidemann[3] has stressed three theories regarding thrombus formation: (1) chemical processes in the blood; (2) mechanical processes in the blood and the vascular system; (3) neurological influences on the entire vascular apparatus. A slight shift in the acidity of the blood aids the ionization of calcium, which activates thrombokinase, leading to more rapid clotting of the blood; on the other hand, a slight shift to alkalinity retards ionization of the calcium and decreases the time of clotting. Trypsin from the pancreas activates prothrombin to thrombin as readily as does thrombokinase, if the calcium ions are available. Mercurial diuretics tend to have the same effect.

The mechanical form of clotting is brought about by the precipitation of fibrin and its collection around solid elements in the blood. Retarded circulation by vein constriction, varicosities near the surface of the body without muscle support, increase in the viscosity of the blood as a result of vasoconstriction[4], and an associated increase in the hematocrit[5] all predispose to clotting. Toxic endothelial damage to the venous walls, and diseases associated with an increased sedimentation rate tend to favor the formation of venous thrombosis.

The effect of the sympathetic nervous system on the metabolism of the body, causing a shift in the acidity of the blood which results in the increased ionization of calcium, predisposes to thrombus formation. The sympathico drugs such as acetylcholine and neostigmine will cause marked fluctuations in the prothrombin index.

Blood pressure changes as regulated by the nervous system, with a resultant slowing of the blood stream and a loss of muscle tone, predispose to clotting. The relationship of infection to venous thrombosis is elusive, but there seems to be real evidence [6] that the clotting factors in the blood may be changed by infection. The use of antibiotic therapy now arises as a possible factor in the process of increased venous thrombosis[7].

Incidence and Diagnosis

The diagnosis of phlebothrombosis is not always easy, and unfortunately a pulmonary infarct or massive pulmonary embolus frequently occurs before complication is detected. Statistics have substantiated the high incidence of pulmonary infarction as the "warning sign" of venous thrombosis. At the University of Pennsylvania during a five and a four year period, the incidence of pulmonary embolism as the "warning sign" was 71 per cent and 81 per cent respectively[8].

Postmortem studies in unselected cases and those in which death was due to secondary massive pulmonary emboli, revealed some discrepancy in the incidence of location of the primary thrombus formation. The incidence appears, however, to be highest in the veins of the lower leg, diminishing in the thigh, and lowest in the pelvic veins. Considering the high incidence of pulmonary embolism as the "warning sign," I feel that the pelvic and thigh veins are involved primarily in a larger number of instances than we expect[9].

Whether the increase be real or apparent, the physician must continue to be vigilant. Daily examination for tenderness over the lateral aspect of the soles of the feet, tenderness and pain in the deep calf muscles elicited either by direct deep pressure in the calf or by lateral compression between the fingers, tenseness of the calf muscles and subcutaneous tissue, the rather sudden appearance of dilated pretibial veins, tenderness behind the head of the fibula, popliteal space, and Hunter's canal will enable us to detect phlebothrombosis earlier. Homan's sign may also be positive.

The diagnosis of thrombophlebitis of the leg in contradistinction to phlebothrombosis is relatively much easier. In the acute thrombophlebitic leg we may find swelling, fever (systemic and local), local increase in heat, painful arteriospasm and discoloration;

whereas, in phlebothrombosis we may find a subtle and many times an asymptomatic process.

Roe and Goldthwait[10] report the incidence of massive pulmonary infarctions in 2 series of autopsies as 1.9 per cent and 2.7 per cent. Each series averaged 1,900 autopsies. DeCamp, Ochsner, and DeBakey[11] reported 35 cases of venous thrombosis in children under 12 years of age during a 12-year period. It was determined that the incidence was 12.4 times greater in the adult than in the pediatric group, but that the mortality rate was 6.1 times greater in the children. Venous thrombosis, though, is rare in children without some major precipitating factor such as a severe infection, marked dehydration, and inanition.

Venous thrombosis is seen in many diseases, the incidence being quite high in heart disease. In one series, 22 per cent of the patients with congestive heart disease had pulmonary infarcts[12]. At Charity Hospital, 54 per cent of the patients with fatal pulmonary emboli were medical patients.

Prevention

Because of the high and apparently increasing incidence, we must endeavor to prevent, diagnose early, and adequately treat this complication. The following are suggestions for lessening the incidence of phlebothrombosis and pulmonary embolism.

1. During surgical procedures place the legs on a soft surface, with the weight evenly distributed.
2. Avoid as much wound trauma as possible.
3. Prevent hemoconcentration and retardation of flow by maintaining fluid and electrolyte balance.
4. Prevent the extremities from becoming wet and cold.
5. Avoid positions that predispose to venous stasis.
6. Provide for activity of the lower extremities during bed rest.
7. Assure early ambulation, provided it is not a static vertical position.
8. Prescribe deep breathing in bed to aid emptying of the veins.
9. Avoid the use of tight abdominal binders and provide relief from abdominal distention.
10. Use anticoagulants as a prophylaxis in selected cases.

Treatment

There are and have been two schools of thought on the treatment of this complication one advocating venous ligation and the other the use of anticoagulants. Many have considered the two methods as rivals, but I feel that they should be considered as complementary. A review of the literature indicates a trend towards anticoagulant therapy, with evidence of much greater faith in the various anticoagulant drugs now than formerly. Many still prefer superficial femoral or vena caval ligations to anticoagulant therapy. Evidence indicates, however, that vein ligation is not an adequate answer to the problem of preventing pulmonary embolism[13].

Venous ligation

Some of the indications for venous ligation are: (1) thrombophlebitis preoperatively or antepartum; (2) after brain and spinal cord surgery; (3) after operations leaving large denuded areas; (4) hepatic or renal insufficiency[13a]. There are some who feel that anticoagulant therapy has supplanted venous ligation as the treatment of choice in all the conditions given except hepatic or renal insufficiency.

Erb and Schumann[14] report a series of 100 cases of fracture of the neck of the femur in which they performed 50 bilateral superficial femoral ligations, using the remaining 50 cases as a control. They had 29 deaths in each group. No anticoagulant therapy was given either before or after surgery and none in the control group.

Roe and Goldthwait[10] found in 1929 femoral vein ligations that the incidence of massive pulmonary embolism was 3.5 per cent. It was concluded after the five-year period of evaluation that the large-scale program of femoral vein ligation had failed to alter significantly the incidence of mortality from pulmonary embolism. However, they felt that certain selected cases would show more favorable results with prophylactic ligation.

Baker and his group[13b], in evaluating the use of anticoagulants and femoral vein ligation, concluded that venous ligation is more useful as a protection against massive than multiple emboli, and that one cannot conclude that prophylactic ligation of the femoral vein has any decisive influence in preventing pulmonary embolism.

I have felt for some time that ligation

should be reserved for patients in the following categories: (1) those having a pulmonary infarction while on anticoagulant therapy, provided the levels of the clotting or prothrombin times have been maintained at the proper therapeutic levels; (2) any patient demonstrating a resistance to heparin and Dicumarol; (3) patients with hemorrhagic diseases; (4) those having severe hepatic or renal insufficiency.

Anticoagulant therapy

There are on the market today numerous anticoagulant drugs, such as various forms of heparin, which prolong the clotting time, and prothrombin depressing drugs such as Dicumarol, Tromexan, phenylinanedione and Compound 63. The most commonly used agent is the combination of heparin and Dicumarol, the heparin being used for its immediate effect, and to protect the patient until a satisfactory prothrombin time level has been obtained.

Loewe[15] has shown that heparin actually causes the "sludge" type of clot to disappear. Accumulated statistics now show that subsequent emboli do not occur and that metastasis of the bland phlebothrombosis clot comes from new unrecognized foci and rarely from clinically recognized phlebothrombosis. By heparinization of the patient, new foci are prevented from forming[16].

The advent of heparin in menstruums has made anticoagulant therapy more popular, safe, and reliable. Nevertheless, there is still some danger if the drugs are not adequately controlled by properly ascertained coagulation and prothrombin times. Protamine sulfate is valuable as a rapid antagonist to heparin. Vitamin K_1 given intravenously as an emulsion returns an elevated prothrombin time to normal or near normal in six hours without a subsequent rebound of the prothrombin time. It may be necessary on occasion to repeat the dose in 6 to 12 hours[17].

Statistically, anticoagulant therapy is as safe, if not safer than venous ligation in the majority of the cases, and has several other advantages such as preventing arterial thrombosis and gangrene in arteriosclerotic patients, inhibiting the spreading of venous thrombosis in systemic veins and in pulmonary arteries if a benign embolus has already occurred, and decreasing the incidence of the postphlebitic syndrome[18].

Summary

It is evident from the high incidence of phlebothrombosis and pulmonary embolism that we must make further strides in securing diagnostic tests which will enable us to make the diagnosis earlier and possibly predict its occurrence in certain individuals.

Kay[19], in his work with alpha-tocopherol, has come to the conclusion that antithrombin levels of 1:8 in combination with normal or near normal prothrombin times indicate a prethrombotic state. The prothrombin activity[20] and antithrombin level determinations are definitely an advance towards effective prophylactic control of pulmonary embolism.

The use of anticoagulant therapy has, in the majority of cases, become the method of choice as a result of its effectiveness and a better understanding of its administration and control. The advent of heparin in menstruum has made its use safer and easier.

Dicumarol is considered safer now as a result of Vitamin K_1 to combat effectively its hypoprothrombinemic effect. It is too early to evaluate the effectiveness and safety of the newer anticoagulant drugs.

Venous ligation should be reserved for patients demonstrating hepatic and renal insufficiency and for other selected cases.

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THE USE OF PYRAHEXYL IN THE TREATMENT OF ALCOHOLIC AND DRUG WITHDRAWAL CONDITIONS

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The use of cannabis, or marihuana as we best know it, and related compounds antedates recorded history. Its earliest use was in Oriental countries, and it was introduced into Western medicine about the middle of the nineteenth century. In reading through the literature one would gain the impression that marihuana-like compounds have been used mainly as intoxicants. Actually this group of drugs has been used legitimately for years in the treatment of various bodily complaints and conditions. For example, Dr. Oscar W. Bethea, in his book, *Practical Materia Medica and Prescription Writing*[1], published in 1917, listed several prescriptions using cannabis in some form. He mentioned the use of the drug as a sedative, an anodyne, and a narcotic, and suggested its use in the treatment of headache, neuralgia, and kindred conditions. It is unfortunate that through the recent exposes in the popular press marihuana has gained such a bad name.

A few years ago Professor Roger Adams, at the University of Illinois, developed synthetic substances resembling marihuana to be used in the study of the marihuana problem. One of these has been called Pyrahexyl in this country and Synhexyl in England. We shall not attempt in this clinical paper to go into the various chemical and pharmacologic aspects of marihuana in any detail. Those who are interested, may refer to Professor Adams' report, appearing in the 1941-1942 edition of the Harvey Lectures[2]. The first comprehensive study done on Pyrahexyl compound was done by Edwin G. Williams and others at the U. S. Public Service Hospital, Lexington, Kentucky[3].

Effects

Briefly, marihuana produces in the majority of individuals general impairment of cerebral function, with mild clouding of consciousness. The higher physiologic functions —those mediated by the thalamus and the cortex in their normal interrelationship — are the most easily disturbed by the drug. Most observers agree that its central action far outweighs any peripheral effects. It acts predominantly on the higher functions. It appears to impair the activity in most areas of the cortex, although those functions which are commonly localized in the frontal lobe appear to be the most acutely affected.

The observed physical effects are (1) elevation of the pulse rate in direct proportion to the degree of intoxication by the drug; (2) elevation of the blood pressure, varying with the individual but usually rising in direct proportion to the pulse; (3) injection of the conjunctival blood vessels, varying with the dose; (4) dilatation of the pupils and sluggish reaction to light and accommodation; (6) slight change in the vision for proximity, distance, and color; (5) circumoral tremors, with tremulousness of the protruded tongue and the extremities; (7) dryness of the oral and pharyngeal mucous membranes; (8) increased frequency and decreased amplitude of thoracic respiratory movements; and (9) ataxia with hyperreflexia. Not all of these effects occur in all individuals, but one or more will be present.

The observed psychologic effects are (1) apprehension and anxiety; (2) euphoria; (3) loquaciousness; (4) lowering of inhibitions; (5) hunger and thirst; (6) feeling of being "high"; (7) uncontrollable bursts of laughter or giggles; and (8) drowsiness, languor, lassitude, and a pleasant feeling of fatigue. Clinical tests reveal that marihuana produces no significant changes in basal

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metabolic rates, blood chemistry, hematologic picture, liver function, kidney function, or electrocardiographic studies. Marihuana delays gastric and intestinal motility somewhat, and produces a definite increase in the frequency of the alpha wave in electroencephalographic recordings, thus indicating increased relaxation.

In recent years conflicting reports have appeared in the literature concerning the use of marihuana-like drugs in treating the opiate withdrawal syndrome. Allentuck and Bowman found that with the marihuana substitution method of treatment the withdrawal symptoms were ameliorated sooner[4]. Yet Himmelsbach states that a synthetic marihuana-like drug had no appreciable ameliorative effect on the opiate abstinence syndrome[5]. The dosage used in both case studies was approximately the same.

More recently, work has been done in England by various investigators. Parker and Wrigley reported a total of 62 patients, indicating psychotics of various types as well as depressive states, in which they recorded subjective improvement in cases of melancholia and neurotic depression. However, when an inert drug was given to a control group, similar results were obtained[6].

Material, Method, and Response

Late in 1949 we were able to secure a supply of Pyrahexyl* for clinical investigation. In the intervening years we have used the drug in 101 cases. Originally, because of the pharmacologic properties of the drug, which usually produce exaltation and feellings of happiness, and because of the reports in the British literature, we used the drug in treating patients with symptoms of depression. Then, because of results reported by Terry from the Livermore Sanitarium in California[7], we began to use Pyrahexyl in the treatment and management of acute alcoholic withdrawal symptoms and those associated with the withdrawal of various addicting and habituating drugs.

Depressive states

Our method of treatment for the cases of depression was, briefly, as follows: Three 15 mg. doses of Pyrahexyl were administered to the patient the first day of admission. If there was no alleviation of symptoms, the dosage was raised by 15 mg. daily, either until improvement resulted or toxic symptoms appeared. The latter included nausea, "giddiness," disturbance in space and time relationships, and other signs which will be mentioned later. The results obtained showed that the drug, in our opinion, was not of sufficient benefit to warrant continuation of this phase of the study. In 20 cases of neurotic depression, only 4 showed evidence of improvement. In the 6 cases of psychotic depression treated we found no improvement. Because of these disappointing results, the use of Pyrahexyl in the treatment of depressive states was abandoned.*

Alcoholic and drug addiction states

The major part of this report is based on the results obtained in the 70 post-alcoholic states and the following drug withdrawal cases: barbiturates 6; morphine 6; Dilaudid 4; Demerol 12; Pantopon 2; and paregoric 1. The most gratifying results were obtained in the 70 post-alcoholic cases. The method of treatment was, in the main, similar to that previously outlined for use in the depressive states.

Most of the patients were admitted to Graylyn in the typical post-alcoholic state. The symptoms presented were tremulousness, restlessness, apprehension, sleeplessness, and anorexia. Their mood was irritable and depressed, and they were unable to sit still. In many instances the patient had eaten little for days prior to admission. Immediately on admission and after a physical examination was done, the patient was given 15 mg. (1 capsule) of Pyrahexyl. This dose was repeated twice on the day of admission. Fifteen milligrams 3 times daily was continued for from 3 to 5 days.

In a large percentage of cases the patients showed a favorable response to the medication. From 30 minutes to 2 hours after ingestion of the first capsule the patient became calm, lost his irritability and restlessness, began to "feel" better, and developed a good appetite. Following the eating of a meal he generally went to sleep for several hours. With the dosage employed, we encountered only a few side reactions. These were usually very mild and confined to generalized dull, aching headaches, minimal ataxia, and dryness of the mucous membranes. During the three to five days of

*Supplied by Abbott Laboratories, Inc. Thanks are due to R. K. Richards, M.D., director of pharmacologic research, for supplies of the drug.

*Dr. Douglas M. Kelley introduced the use of Pyrahexyl at Graylyn. He started the experiments in depressive reactions, but, as noted, the results were not encouraging.

treatment the patients remained mildly euphoric and happy, their appetite remained good, and they slept well. When the medication was stopped, we noted no withdrawal symptoms. Our experience in this respect paralleled that of Allentuck and Bowman[4], who felt that the use of marihuana does not give rise to a biologic or physiologic dependence, and that discontinuance of the drug does not result in withdrawal symptoms.

Results

In the treatment with Pyrahexyl of 70 cases of the post-alcoholic syndrome we can report clinical alleviation of the symptoms in 59, or 84.28 per cent. The 11 cases that did not show improvement (or 15.72 per cent) did not differ a great deal clinically from the other 59. By that we mean that the duration of the last alcoholic bout was about the same, and the amount of alcohol consumed was no different from that in some patients who did show improvement. The same applied to age and sex incidence. We have no explanation as to why 59 out of 70 patients showed improvement while 11 did not. Perhaps an individual idiosyncrasy to the drug is the explanation, for it is known that individual reaction to other drugs do occur.

In the treatment of drug addictions our experience is less extensive but none the less significant. Out of 6 cases of barbiturate addiction, amelioration of symptoms was noted in 4; in 4 cases of dilaudid addiction we noted alleviation in 3; in 2 cases of pantopon and one of paregoric addiction all patients reported a smooth withdrawal course, without the usual symptoms noted in such conditions. We were especially pleased with the results obtained in 12 cases of Demerol addiction. With 10 of these patients, or 83 per cent, we were able to withdraw the drug completely in one week's time without having to resort to any other type of medication. Occasionally a patient would report mild apprehension or transient cramp-like pains of the lower extremities, but these were infrequent and when they did occur, were mild and transient. The 2 patients who showed no response were long-term users of Demerol who had received treatment on two other occasions by the gradual withdrawal method.

With 6 cases of morphine addiction the results were less satisfying. Only 2 patients reported a course of withdrawal without any markedly unpleasant symptoms with the use of Pyrahexyl. It was necessary in the other 4 patients to use other methods of treatment.

In those cases where good results were obtained by the use of Pyrahexyl in the treatment of drug withdrawal symptoms, the side symptoms produced were of a mild character. The patients felt physically stronger and showed increased psychomotor activity. They had an increased appetite and such withdrawal symptoms as nausea, diarrhea, and perspiration were diminished or eliminated. The feeling of euphoria produced helped in rehabilitating the physical condition and in facilitating social reorientation. An outstanding result was a subjective feeling of relaxation. The sleep induced by the drug likewise contributed to the general improvement in the patient's health.

Comment

At the time of the first examination the patients with post-alcoholic syndromes were beginning to feel a severe "hangover" and subjectively complained of remorse, agitation, depression, tremulousness, and tension. They all complained of anorexia and some of nausea. The administration of Pyrahexyl produced within 30 minutes to 2 hours a marked change in the psychologic and physiologic status of 84 per cent of the patients in our series. With continued administration of adequate doses over a 3 to 5 day period, the patients passed smoothly through their period of alcoholic withdrawal, without marked subjective complaints. From a psychological standpoint, it is our feeling that this result is beneficial in the over-all handling of a patient and in establishing rapport. We do not hold with the opinion that alcoholics must be treated harshly and made to "sweat it out" in order to repent of their actions. Our experience with Pyrahexyl indicates that alcoholic patients appreciate consideration for their feelings and respond to it by being more cooperative in therapeutic relationships.

In those patients who presented complaints resulting from the withdrawal of various drugs (or medicaments)—namely, barbiturates and opiate derivatives—the picture was similar. We attribute the difference in the results obtained in Demerol addicts from others to the individual addictive properties of the drug itself and the development of less physiologic dependence on the drug as compared to other opiate derivatives.

Illustrative Cases

The results of treatment with Pyrahexyl in one patient with drug addiction and in one with alcoholism will be described briefly:

Drug addiction (Case 1)

A 32 year old man entered Graylyn Hospital with the chief complaint of morphine addiction for the past one and one-half to two years, and of pain in both ankles of about two years' duration. He was admitted to the hospital for insulin subshock therapy and for the treatment of morphine addiction. He had been taking from ¼ to ½ grain intramuscularly every 4 hours for a long time prior to entry here. On admission he was extremely lethargic, garrulous, and uncooperative, and complained bitterly of pain in his lower extremities, although there were no positive physical findings referable to this area. He did express a strong desire to be free of drug addiction and to return to normal health again. The physical examination was not remarkable in the main. Laboratory findings were within normal limits.

Following admission to the hospital he was given Pyrahexyl tablets—3 the day of admission, 3 then for the next five days, and 1 on the sixth day. During this period mild euphoria was noted and the patient gradually began to complain less of pain in his legs, was much easier to manage, and cooperated to a much greater extent. He did notice a sensation of elation and at times felt rather dizzy, but experienced no nausea or vomiting or any other side-effects of note. He was not given morphine from the time of entrance. With the administration of Pyrahexyl, paraldehyde, and Sodium Amytal, none was deemed necessary. Throughout his 20-day hospitalization period, during which he received insulin subshock therapy, he did very well and was discharged much improved. He volunteered the information that some type of medicine that we were giving him had made him feel better than anything he had ever used and, as mentioned, he did notice euphoria, elation, and some dizziness.

Alcoholism (Case 2)

A 37 year old married businessman was brought to the hospital by his brother for consultation about over-indulgence in alcohol. When he first entered the office, it was obvious that the patient had had a drink or two on the way down to fortify himself for the ordeal. He was tremulous, somewhat thick in speech, perspiring freely, and quite restless.

The brother revealed a history of excellent business ability on the part of the patient but told also about numerous periods of excessive drinking. The patient readily admitted the former but minimized the latter. During his denial of any problems he grew more restless and pale, and frankly asked for a drink. He was told that patients were not treated with bourbon or scotch, but that we had other methods of treatment. He was given a capsule of Pyrahexyl, and the interview continued. In about 20 minutes the patient spontaneously remarked that he felt better. He was asked whether he still felt the need for a drink and his answer was in the negative. Admission to the hospital was arranged without further argument or persuasion.

On the day of admission he received 2 more Pyrahexyl capsules. On the following day 2 more were given and then this medication was discontinued. The restlessness and tension had subsided and the patient had joined the group activities.

With the aid of psychologic studies and the efforts of the psychiatric social worker in adjusting family problems, the patient returned to his place in business and in the community, where he has remained sober and efficient for almost a year. In the meantime he has referred another member of his family for treatment of alcoholism.

Conclusions

It is our conclusion that Pyrahexyl and related compounds are beneficial in the treatment of withdrawal symptoms from the use of alcohol to a marked degree, and in the treatment of withdrawal symptoms from the use of opiates to a less marked, but still significant degree. We offer for consideration an over-all series of 101 patients in whom Pyrahexyl has been used. We have not been able to differentiate, prior to therapy, those patients who would fall into the group successfully treated from those who would not respond. Perhaps this paper will stimulate some to continue this study with such a view in mind. We offer it as a preliminary report and study.

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Most psychiatry should be done by the patient's own doctor; all that is needed is time, interest and a knowledge of the things that go into making a normal person. We could all do with some instruction in the last-named, incidentally. I keep hoping that some day the psychiatrists will take time out from the ids, the egos and the sex lives and tell us what they've learned about the human emotions and how they grow.—Copping, G. A.: Common Pitfalls in Medical Practice, Canad. M.A.J.: 66:331 (April) 1952.

Provision for individual patient instruction in the significance of asymptomatic tuberculous lesions of small extent, or indeed of all inactive or suspected tuberculosis, is a great need which has grown out of mass survey examinations. This can be provided only by giving both clinic physicians and nurses the time to give each patient the full explanation he deserves.—Howard M. Payne, M.D., Philip Enterline, and Julia Heuck, The Am. Review of TB, Nov., 1952.

MASSIVE GASTROINTESTINAL HEMORRHAGE DUE TO INTESTINAL HEMANGIOMA

With a Case Report

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Massive gastrointestinal hemorrhage is a frequently encountered emergency. The immediate mortality of this condition has been reported in a series given by Wherritt[1] to be as high as 22 per cent. Contributing to this high figure probably is the fact that many of the less severe cases of hemorrhage do not reach the hospitals and thus are not recorded in their statistics. Walters[2] and Meulengracht[3] agree that approximately 90 per cent of the cases of gastrointestinal bleeding are due to peptic ulcers; while Bockus[4] says that the percentage is nearer 60 to 75 per cent. The few remaining cases are due to a wide variety of conditions which can be quite difficult to diagnose and treat.

We have recently had a case due to intestinal hemangioma, which is one of the rare causes of massive gastrointestinal hemorrhage. A brief case report follows.

Case Report

A white man, 58 years of age, was admitted to the Jefferson Hospital through the emergency room on October 22, 1952, in moderately severe shock from loss of blood. He had had tarry stools for two days and had just begun vomiting bright red blood. He had had two previous episodes of melena but no hematemesis. The first episode of tarry stools had begun in December, 1950, and continued for two months thereafter before he consulted his family physician and was hospitalized with a tentative diagnosis of a bleeding peptic ulcer. Ten units of whole blood were necessary to combat his anemia. The bleeding stopped on a medical regimen. Although roentgenograms at that time failed to show an ulcer, the patient was put on an ulcer regimen on the basis of his statement that he had had some mild indigestion, but no pain, all his life. He got along satisfactorily until 10 months later, in December, 1951, when he began to have tarry stools again. He consulted his physician, who placed him on medication and bed rest at home. The bleeding promptly stopped. Roentgen examination again at this time showed no ulcer.

The patient had been doing satisfactorily up until two days before the present admission, when he once again noticed tarry stools. He went to his physician, who prescribed medicine and advised him to go home to bed. He continued to have tarry stools, but did not feel ill. On the day of admission he suddenly started vomiting bright red blood, collapsed, and was brought to the hospital in shock. The blood pressure was 96 systolic, 50 diastolic, the red blood cell count 3,700,000, hemoglobin 76 per cent, urine normal. The patient was alert, and on limited abdominal examination (because of active hemorrhage) no masses or tenderness were noted. One thousand cubic centimeters of whole blood was given immediately and the patient responded well. The bleeding gradually stopped. He received an additional 3,000 cc. of blood over a period of five days to bring his blood count back to normal.

One week after admission the abdominal cavity was entered. Exploration revealed no ulcer of the stomach or of the first and second portions of the duodenum. The stomach was opened to facilitate the examination, and no abnormality was found. Examination of the small intestine revealed a number of hemangiomas 1 to 4 mm. in size over the entire length of the jejunum and ileum, the greater number being in the jejunal segment. None was seen in the stomach or the colon. Four or five of the larger lesions were excised for examination. The patient's postoperative course was uneventful.

Following the operation examination of the patient's skin revealed a number of small 1 to 5 mm. hemangiomas on the right anterior chest wall and an occasional small hemangioma on the back. The pathologic report was multiple capillary hemangiomas of the small bowel. The patient has had no further bleeding episodes since the operation.

Comment

Control of bleeding

The management of acute gastrointestinal hemorrhage is well established, and it suffices to say that the conservative treatment is preferable to surgery, if at all possible. Horsley and Keasbey[5] have emphasized the fact that emergency operations will increase the mortality of these cases. Of course, in continued or massive bleeding, surgery must be resorted to; the surgeon is at times greatly benefited by the knowledge that an emergency gastrointestinal series can give him, although we recognize this knowledge to be incomplete.

Diagnosis

If conservative treatment has controlled the bleeding, then an attempt at diagnosis must be made. If we are to assume that 60 to 90 per cent of these cases are due to ulcers, the roentgenogram, in conjunction with the history, will be of invaluable aid. When peptic ulcer is ruled out, the establishment of a diagnosis may prove a difficult problem. The studies necessary then will cover an extremely wide variety of conditions that have been reported as causes of massive gastrointestinal hemorrhage. Deaver[6] has pointed out that there is a cer-

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tain low percentage of cases for which no cause is ever found.

Gentry and others[7], in an exhaustive review of vascular tumors of the gastrointestinal tract, state that approximately 600 cases have been reported in the medical literature. He classifies the lesions in four groups: (1) telangectasis, (2) capillary hemangiomas, (3) mixed capillary and cavernous hemangiomas and, (4) cavernous hemangiomas. He estimates the incidence of gastrointestinal hemangiomas to be 1 in 14,000. The cavernous hemangioma is the most frequently found vascular lesion in the gastrointestinal tract.

The diagnosis of hemangioma of the bowel is impossible without operative or endoscopic procedure, and then pathologic study is often necessary. Visualization of a gastric hemangioma by Vinson has been reported by Morton and Burgers[8], who removed the lesion surgically. Kaijser[9] has emphasized the possible preoperative diagnostic value of phleboliths demonstrated roentgenographically.

Cavernous (sessile and polypoid) hemangiomas not uncommonly manifest themselves by symptoms of obstruction. We feel that the appearance of hemangiomas on the body surface or about the nasal or oral cavities may be highly suggestive of internal hemangiomas in cases of undetermined gastrointestinal hemorrhage. Our patient presented a number of hemangiomas on the trunk, and Gentry and others[7], in reporting a series of gastrointestinal hemangiomas, referred, in a few of the cases, to other lesions found upon the trunk, about the nose, and within the oral cavity. In the majority of his cases, no reference is made to the presence of hemangiomas elsewhere on the body.

Treatment

Treatment is varied and depends upon the size, position, and number of the lesions. Surgery alone, or in combination with radiation (radon seeds), is used. Sclerosing fluids are considered impractical and unsafe for use in many sites. Multiple lesions present the biggest therapeutic problem; all classes may be multiple.

The following statements by Gentry and colleagues[7] bear direct quotation:

> "Although in some of the cases of gastrointestinal telangectasia death followed a severe hemorrhage (14%), radical extirpation of these lesions is impractical because of their multiplicity and their tendency towards spontaneous regression . . .
>
> "A large majority of the patients with capillary hemangiomata of the gastrointestinal tract experienced symptoms during life (89%); surgical extirpation was undertaken in many (78%); and only a few died as a direct result of these lesions (11%)."

The malignant tumor, although much rarer than the benign tumor, must be kept in mind when a gastrointestinal vascular lesion is encountered. Here the histolic examination may be the only method of differentiation.

Summary

One of the rarer causes of massive gastrointestinal hemorrhage is intestinal hemangiomas.

The finding of small hemangiomas on the body surface may suggest this diagnosis. Patients usually respond to conservative treatment, but surgery may have to be resorted to. A case which was treated surgically is presented.

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Chronic peptic ulcer of the stomach or duodenum is one of the friendlier chronic diseases. It does not increase in size or severity, though it remains for the life of the patient. Its spectacular complications, hemorrhage, obstruction, and perforation, are not as common in the later as in the middle years. The patients do not have more vascular disease than others of their age. Their life expectancy is not lessened. They have long periods of remission. By the time they reach old age the excitants that cause relapses, such as overwork and worry, have faded, and they find relief in simple rules of health, meals at regular hours, adequate rest and exercise.—Monroe, R. T.: The Effect of Aging of Population on General Health Problems, New England J. Med. 249:322 (Aug. 20) 1953.

EARLY NORTH CAROLINA MEDICINE

DR. CHARLES HARRIS (1762-1825)

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The *Medical Repository*, volume 11, for the year 1808, contains an "Account of the successful Extirpation of Tumours from the Neck, stated in a letter from Dr. Charles Harris to Dr. Calvin Jones; communicated by Dr. John C. Osborn to Dr. Miller." Except for Dr. Miller, who was one of the editors of the journal, the physicians are not identified, but the other three were almost certainly North Carolinians. Dr. Calvin Jones of Raleigh, who had served as corresponding secretary of the state medical society, had contributed to the same issue of the *Repository*, in a letter, an account of Dr. Sterling Wheaton's method of making quicksilver ointment; Dr. John C. Osborn, of New Bern, had been listed as one of the first subscribers to the journal, in 1798[1], had practiced in North Carolina from 1787 to 1807, had been president of the medical society from 1801 to 1804, and in 1807 had moved to New York City, where he had a large practice, and where, as Thacher stated, "He was created Professor of the Institutes of Medicine, in the Medical Faculty of Columbia College, and upon the union of that Faculty with the College of Physicians and Surgeons, he was appointed Professor of Obstetrics and the diseases of women and children."[2]

Dr. Charles Harris of Cabarrus County was at that time well known as a surgeon, and he was apparently the author of the letter, which contains one of the earliest accounts of successful operations of this type to be published in America. The Raleigh *Minerva*, a weekly newspaper, in the issue of May 19, 1808, after mentioning Dr. Osborn's appointment, continued: "The last number of the Medical Repository (from which the above was taken) contains among other communications from physicians of this state, one from Dr. Charles Harris, of Cabarrus, giving an account of his having with complete success operated on three cases of goitre or bronchocele, a disease which has heretofore been deemed incurable."[3]

Wheeler's *Historical Sketches of North Carolina* includes in the account of Cabarrus county a short biographical article on Dr. Harris, who was born in 1762, and who served as a very young soldier under Col. Davie in the later years of the Revolution. After the war, he resumed his studies at Clio Academy, in Iredell county, and later studied medicine, first under Dr. Isaac Alexander at Camden, South Carolina, and then at the University of Pennsylvania. Dr. Harris first practiced at Salisbury and then settled at Favoni, his home in Cabarrus county. His skill and success soon gave him considerable local fame, and he became active as a teacher as well as a practitioner. According to one historian, "He was offered the chair of surgery in the University of Pennsylvania, but declined the flattering offer."[4]. He died in 1825, and the *Catawba Journal*, a newspaper then published in Charlotte, printed a brief obituary notice in its issue of October 4, 1825. After saying that "he was one of our first Physicians, and as a Surgeon, he was unequalled in the western part of this state,"[5] the notice stated that several Charlotte physicians had held a meeting at which they not only passed resolutions of respect, but decided to observe a formal mourning period of forty days in his honor. The inscription on his tombstone, quoted by Wheeler, says that Dr. Harris "engaged in the practice of medicine and surgery forty years; eminent in the former, in the latter preeminent. He was a man of extensive reading, of an acute, inquisitive mind, friendly to all and beloved by all. His heart entered deeply into the sufferings of his patients, mingling the medicine he administered with the feeling of a friend."[6]

The article in the *Medical Repository* consists largely of two case reports, but includes also the surgeon's reasons for deciding to attempt the operation after a period of waiting for a favorable opportunity, and concludes with an expression of his happiness in the successful outcome. His use of the word *bronchocele* in the first paragraph inspired an editorial note that:

"The use of the term Bronchocele is too indefinite, as it is employed by many surgeons to express every indolent tumour which occupies the fore part of the neck. Encysted tumours, of course, come under this latitude of definition, and such seem to have been the nature of the swellings which were so successfully extirpated by Dr. Harris. They certainly were not enlargements of the thyroid gland. Because the attempt to extirpate this, on account of its connection, and the size of its blood vessels, has

*Reference Librarian, Division of Health Affairs, University of North Carolina, North Carolina Memorial Hospital, Chapel Hill.

not only always been held to be hazardous, but in several instances has indubitably proved fatal."

Dr. Harris' letter, with a few omissions, follows:

"I do my self the pleasure of stating to you an operation of surgery, which I lately performed on a lady for Bronchocele; and as an attempt of this kind has hitherto been rare in the annals of surgery, I feel myself under obligations to make you acquainted with the reasons which decided me to hazard the undertaking.

"I have frequently, in the line of my business, been applied to for advice in similar cases; all of them of enormous size, and of so many years standing, that I could not dare recommend anything further than to beguile the time and soothe the mind of the patient; but in which I could make no rational calculations to dissipate or discuss the tumour. The disease proceeded until the patient died, most commonly of compression and stricture of the throat and windpipe, and of varicose distention of the blood vessels of the brain from obstruction of the jugular veins.

"These observations produced a determination in my mind to hazard an operation for extirpation of the tumour on the first willing subject, where the health of the patient was otherwise favorable to calculate a successful cure.

"About three years ago such a case was presented to me. After acquainting the patient of the fatal tendency of the disease, when suffered to go on in the ordinary way, and of the probability of success that extirpation would afford her, she agreed to run the risque. It was of recent origin, not more than a year from the time she had first discovered it, and not quite so large as a pullet's egg. I made an incision longitudinally its whole length through the integument, and then carefully insinuated my fingers between the interstices of the muscles until I had the tumour bare. With my blunt probe conductor and my finger, I found it, as I had apprehended, encysted. I then separated the superincumbent muscles from the tumour, until I had it detached from everything except the trachea: to this I found it firmly adhered by an expansion of a ligamentous-like substance, which proceeded in a tendinous form from the os hyoides. This I dissected from the trachea, beginning at the inferior point, where I found the detachment most easily made, and proceeding upwards, denuding the trachea with a scalpel until I came to the upper extremity, and then finished the operation by dividing the ligament which proceeded from the os hyoides. This appeared of a firm condensed tendinous texture; and it is from hence that I suppose the tumour originated.

"The haemorrhage was very small, so that I did not think it necessary to join the edges of the wound by sutures. An adhesive plaster was applied, and in the course of a few days the edges adhered together by first intention. . .

"About three months since an application was made to me by a lady from South Carolina. I think the bronchocele was fully as large as any I had ever seen; the tumour had been increasing for twenty-two years: it extended from the chin (which it buoyed up) along the trachea, until it descended an inch or perhaps more, under the breast bone, and spread laterally a medial distance to each ear. . .

"I felt some time reluctant to hazard an operation which appeared so truly tremendous. Its magnitude, local situation, and want of precedents deterred me for some time; until the earnest entreaties of the woman at length prevailed, who told me, that even death in the operation could shorten her life but for a few days, as without relief she must soon die a death of suffocation.

"I divided the integument from the chin to the sternum, then penetrated through the interstices of the muscles with my knife and finger, until the tumour appeared; the adhesions were gradually detached, by insinuating my fingers upwards and downwards and laterally, until all the anterior parts were detached, which was not a little difficult, as it was pressed in between the muscles to such an extent that the cellular membrane had become condensed and firm, and more especially on the trachea.

"When I had proceeded thus far, I was reduced to a new difficulty, to get it pressed out of the aperture; for the extremities of the incision were bordered by unyielding abutments, the chin and the breast bone; however, after a few efforts, I squeezed it into an elongated form, and pressed it out. I was under the necessity, as formerly, to use the scalpel to assist in dividing it from the trachea. I found it, also, as the other was, attached to the os hyoides by a large tendinous-like substance, which spread over the whole posterior part of it. This ligament was secured by a ligature before it was divided, lest there might be blood vessels in it, which had given the tumour nutrition; two small arteries were divided and secured when the incision was made through the muscles which covered them. Five interrupted sutures closed the wound. Absorption and adhesion took place in about fifteen days; and about a week afterwards, the patient took her leave under good prospects of complete restoration of health and relief from pain.

"Thus I am happy in having it in my power to assure you that this disease, which hath hitherto appalled surgical aid, admits of complete relief in almost any stage of it while the constitution remains sound. And this is the more important, as this disease is painful from its commencement, and operates as a perpetual sedative to hope by the presence of death visibly and daily making his encroachments, ready to seize the unfortunate victim by the throat and extinguish life. . .

"There is every reason that humanity can urge for surgeons to make themselves acquainted with the real nature and history of the disease in question, as far as it is in their power to do it; in order to relieve and thereby continue the blessings of life and health to many of our fellow creatures who have hitherto been consigned to no other alternative than the grave."(7)

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1. Medical Repository 1:15, 1798.
2. Thacher, J.: American Medical Biography, Boston, Richardson & Lord, 1828, p. 415.
3. Minerva. Fayetteville and Raleigh. May 19, 1808.
4. Alexander, J. B.: The History of Mecklenburg County from 1740 to 1900, Charlotte, 1902, p. 185.
5. Catawba Journal, Charlotte and Salisbury, October 4, 1825.
6. Wheeler, J. H.: Historical Sketches of North Carolina from 1584 to 1851 . . . New York, Hitchcock, 1925, v. 2, p. 68 (Reprint of the original edition as written in 1851)
7. Medical Repository 11:246-249, 1808.

The decline in the annual number of new cases of tuberculosis reported during recent years has been slight compared with the decline in mortality. That the number of newly reported cases remains high, despite the rapid decline in death rates, underscores the fact that efforts to wipe out tuberculosis must continue to have high priority among public health problems.—Division of Chronic Disease and Tuberculosis, Public Health Service, Public Health Reports, June, 1953.

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"The prime object of the medical profession is to render service to humanity; reward or financial gain is a subordinate consideration. Whoever chooses this profession assumes the obligation to conduct himself in accord with its ideals."—Principles of Medical Ethics of the American Medical Association, Chapter 1, Section 1.

OCTOBER, 1953

THE CORNELL CRASH INJURY RESEARCH PROGRAM

For many years the slaughter on the nation's highways has been deplored, and various suggestions have been made for decreasing highway accidents — such as requiring that all cars be equipped with governors to regulate their speed, increasing the age limit for granting drivers' licenses, and giving jail sentences for drunken driving.

In 1950 the Indiana State Police began to study the factors involved in highway accidents. The study has attracted nation-wide interest. Since 1942 the department of Public Health and Preventive Medicine at Cornell, under the sponsorship of the Army, Navy, Air Force, and the Civil Aeronautics Administration, had been conducting a study of the factors involved in aviation crash injuries. Last year the Indiana program and the Cornell Crash Injury Research Project began to coordinate their activities, with Cornell reviewing the data obtained by the Indiana State Police.

In the study an entirely new viewpoint has evolved. It was recognized that, human nature being what it is, crashes will occur as long as vehicles are driven or piloted by human beings, but that it should be possible to make the inevitable accidents productive of fewer crippling or fatal injuries. To this end, highway accidents are to be studied with a view to learning how to build more safety factors into automobiles.

It was decided that best results could be obtained by an intensive study of small sampling areas, rather than a widespread national survey. North Carolina has been selected for a pilot study. (Incidentally, this is the third time that North Carolina has been chosen to blaze the trail in a nation-wide research program.) The fact that Mr. John O. Moore, who is doing the field work for the Cornell Crash Injury Research Project, is a native son of North Carolina may have influenced the choice; but the excellent reputation of our State Public Health Department and of our Highway Department also helped.

The Executive Council of the State Medical Society, at its regular meeting in Raleigh on September 27, voted unanimously to endorse the study. It is a foregone conclusion that North Carolina doctors will back the program, which is outlined in the following statement presented by Mr. Moore to the Council.

* * *

STATE OF NORTH CAROLINIA

PROPOSED AUTOMOBILE CRASH INJURY RESEARCH PROGRAM

JOINTLY UNDERTAKEN BY
THE DEPARTMENT OF MOTOR VEHICLES
AND
THE DEPARTMENT OF PUBLIC HEALTH
IN COLLABORATION WITH
CORNELL UNIVERSITY MEDICAL COLLEGE

In a further effort to reduce the injury and deaths on North Carolina highways, the Department of Motor Vehicles and Department of Public Health have undertaken a re-

search program in collaboration with Cornell University Medical College's Crash Injury Research Project. The primary purpose of the program will be to gather information on the basic causes of human injury in automobile accidents occurring on the rural highways of the state. The program will have these four basic aims:

1. To provide factual information

2. To analyze accident data, in order to assess current dangers and safety needs, from the injury-producing property of automobiles presently in use

3. To obtain field reports which may be used to evaluate the result of engineering design and the changes needed to modify the exposure to injury in automobile accidents

4. To stimulate engineering research and safety design studies by university and non-automotive groups.

The mechanics of the proposed program are as follows:

1. The state highway patrolmen will collect "on the scene accident data" on all fatal and personal injury accidents in selected sampling areas, and will photograph interior and exterior structural damage of the automobiles involved in accidents.

2. In the sampling areas, the state highway patrolman will be responsible for reporting automobile accidents to the local health officer, stating the number of persons involved and the number injured.

3. The state highway patrolman will then forward a completed Cornell Accident Report Form CCIR-AU-2, and accident photographs, along with the official State Accident Form No. 349 to the Accident Records Division, Department of Motor Vehicles. In cases where photographs are not available for forwarding with the Cornell CCIR-AU-2 Form; the photographs will be forwarded at a later date, with a coding which will allow the photographs to be correlated with the correct accident form.

4. The Accident Records Division will screen reports for completeness, and will then correlate the accident form with the photographs and forward both items to the State Department of Public Health.

5. The State Department of Public Health will be responsible for completing Cornell University Medical Form CCIR-AU-1 with information obtained from the local health officer with regard to all individuals injured in the accident and will assemble the correct medical forms and accident form and photographs. These will be forwarded once or twice a month to the Cornell Medical College Crash Injury Research Project.

6. The Crash Injury Research Group of Cornell will return to the Department of Public Health and the Department of Motor Vehicles IBM card decks punched on all accidents submitted by the State of North Carolina. These decks may be used for research purpose by the two departments.

7. Cornell University Medical College Crash Injury Research Project personnel will assist the Department of Motor Vehicles and the Department of Public Health in training patrolmen and health officers in the sample areas, if such assistance is requested.

8. Sample areas will be selected jointly by the Department of Motor Vehicles and Department of Public Health and used for periods not longer than two months.

9. Cornell University Medical College Crash Injury Research Project will furnish without cost to the State of North Carolina necessary numbers of accident and medical forms for use in the proposed program.

* * *

FIFTIETH ANNIVERSARY OF NEW JERSEY'S MEDICAL JOURNAL

The *Journal of the Medical Society of New Jersey* began its fiftieth year of publication with the September issue. The anniversary is duly commemorated by a special issue, with the cover a gleaming gold in color. The leading editorial is devoted to a sketch of the *Journal* and of New Jersey medicine. The second editorial, contributed by a former editor, Dr. Henry A. Davidson, has the intriguing title, "Ours Is a Comfortable Journal." Dr. Davidson makes a good case for this theme.

The original articles all deal with various phases of medicine, especially as they pertain to New Jersey, during the past fifty years.

The NORTH CAROLINA MEDICAL JOURNAL, now in its fourteenth year, offers hearty congratulations to its much older brother, and best wishes for its continued success.

POSTOPERATIVE STAPHYLOCOCCAL INFECTION FOLLOWING ANTIBIOTIC THERAPY

A case recently presented in a clinicopathologic conference of the Massachusetts General Hospital[1] brings forcibly to attention an observation that is being made with increasing frequency — that the use of the modern antibiotic is not without danger. A patient with polyps of the sigmoid had been prepared for resection with Sulfathalidine for four days before the operation. Postoperatively he was given streptomycin and penicillin. After the operation the patient began to have diarrhea and a high fever. He died on the sixth postoperative day. Autopsy revealed membranous enteritis and generalized staphylococcal septicemia.

In the discussion, it was stated that this was the eighth case of pseudomembranous ulcerative enteritis that had occurred in the Massachusetts General Hospital within the past two or three years. Five of these patients died.

The multiplication of such cases will cause many physicians to share the view expressed by Dr. Thomas F. Paine in the discussion: "If I had to face a bowel resection today I think I should prefer to face it with the organisms I am ordinarily used to having in my bowel rather than having a new species selected out through no choice of mine. Staphylococcal enteritis is one of the complications of antibiotic therapy."

1. Case Records of the Massachusetts General Hospital, Case 39271, New England J. Med. 249:29-33 (July 2) 1953.

MECKLENBURG COUNTY'S CONTRIBUTION TO PUBLIC RELATIONS

In the Organization Section of the *Journal of the American Medical Association* for July 18[1], four and one-half pages are devoted to a report on an "Opinion Survey and Inventory of Medical Care, Personnel, and Facilities in Mecklenburg County, N. C." The survey was made by a special committee appointed early in 1951 by Dr. Monroe Gilmour, who was then president of the Mecklenburg County Medical Society. Dr. David Welton was chairman of the committee. Other members were "Dr. William T. Raby, vice Chairman; Dr. Andrew Taylor, Secretary; 1951, Dr. Gregg Watters; 1952, Dr. Addison Brenizer, Jr., Research; Dr. Ralph Bell, Physician Personnel; Dr. Z. Vance Kendrick, Charlotte Dental Sociey; Miss Ann Rhodes, District Nurses Association; Dr. M. B. Bethel, City and County Health Services; Dr. Rudolph M. Wyche, Negro Physicians and Dentists; Mr. Marshall Pickens, Hospital Facilities; and Mr. Hoyt Galvin, the Public. The Advisory Group were Drs. Hamilton McKay, V. K. Hart, Luther Kelly, H. L. Newton, Monroe T. Gilmour, ex-officio, 1951, and E. R. Hipp, ex-officio, 1952."

The committee did an enormous amount of work in making and tabulating the survey. Since most readers of the NORTH CAROLINA MEDICAL JOURNAL also get the J.A.M.A. the report will not be quoted in full—but it may be said that it was on the whole favorable to the medical, dental, and nursing professions. The purpose of this brief editorial is two-fold: (1) to call to the attention of our readers to the full report in the J.A.M.A.; (2) to congratulate Dr. Welton, the committee, and the Mecklenburg County Medical Society on their splendid contribution to medical public relations. Other communities might well follow this example.

1. J.A.M.A 152:1145-1149 (July 18) 1953.

THE 1953 ROSTER

More and more North Carolina physicians are finding a valuable aid in the Roster of Fellows of the Medical Society of the State of North Carolina, which is published annually as a supplement to the August issue of the NORTH CAROLINA MEDICAL JOURNAL. Like its predecessors, the 1953 edition contains a list of the officers and committees of the Society, an alphabetical list of Fellows with business and post office addresses, and a roster by counties. At the suggestion of several members a new feature was added this year — the inclusion of business telephone numbers immediately preceding the addresses in the alphabetical list of Fellows. This item should prove a time-saver to hard pressed physicians.

Those who have not yet made use of the Roster as a ready source of information concerning their fellow physicians should lose no further time in doing so.

Committees and Organizations

BOARD OF MEDICAL EXAMINERS OF THE STATE OF NORTH CAROLINA

MEDICAL PRACTICE ACT

WITH 1953 AMENDMENTS

Art. 1. Practice of Medicine.

§ 90-1. North Carolina medical society incorporated.—The association of regularly graduated physicians, calling themselves the state medical society, is hereby declared to be a body politic and corporate, to be known and distinguished by the name of The Medical Society of the State of North Carolina. (Rev., s. 4491; Code, s. 3121; 1858-9, c. 258, s. 1; C. S. 6605.)

§ 90-2. Board of examiners.—In order to properly regulate the practice of medicine and surgery, there shall be established a board of regularly graduated physicians, to be known by the title of The Board of Medical Examiners of the State of North Carolina, which shall consist of seven regularly graduated physicians. (Rev., s. 4492; Code, s. 3123; 1858-9, c. 258, ss. 3, 4; Ex. Sess. 1921, c. 44, s. 1; C. S. 6606.)

§ 90-3. Medical society appoints board.—The medical society shall have power to appoint the board of medical examiners. (Rev., s. 4493; Code, s. 3126; 1858-9, c. 258, s. 9; C. S. 6607.)

§ 90-4. Board elects officers and fills vacancies.—The board of medical examiners is authorized to elect all such officers and to frame all such by-laws as may be necessary, and in the event of any vacancy by death, resignation, or otherwise, of any member of said board, the board, or a quorum thereof, is empowered to fill such vacancy. (Rev., s. 4494; Code, s. 3128; 1858-9, c. 258, s. 11; C. S. 6608.)

§ 90-5. Meetings of board.—The board of medical examiners may assemble once in every year in the city of Raleigh, and shall remain in session from day to day until all applicants who may present themselves for examination within the first two days of this meeting have been examined and disposed of; other meetings in each year may be held at some suitable point in the state if deemed advisable. (Rev., s. 4495; 1915, c. 220, s. 1; 1935, c. 363; C. S. 6609.)

§ 90-6. Regulations governing applicants for license, examinations, etc.—The board of medical examiners is empowered to prescribe such regulations as it may deem proper, governing applicants for license, admission to examinations, the conduct of applicants during examinations, and the conduct of examinations proper. (1921, c. 47, s. 5; Ex. Sess. 1921, c. 44, s. 2; C. S. 6610.)

§ 90-7. Bond of secretary.—The secretary of the board of medical examiners shall give bond with good surety, to the president of the board, for the safe-keeping and proper payment of all moneys that may come into his hands. (Rev., s. 4497; Code, s. 3134; 1858-9, c. 258, s. 17; C. S. 6611.)

§ 90-8. Officers may swear applicants and summon witnesses.—The president and secretary of the board of medical examiners of this state shall have power to administer oaths to all persons who may apply for examination before the board, or to any other persons deemed necessary in connection with performing the duties of the board as imposed by law. The board shall have power to summon any witnesses deemed necessary to testify under oath in connection with any cause to be heard before it; or to summon any licentiate against whom charges are preferred in writing, and the failure of the licentiate, against whom charges are preferred, to appear at the stated time and place to answer to the charges, after due notice or summons has been served in writing, shall be deemed a waiver of his right to said hearing, as provided in § 90-14.2 (1913, c. 20, s. 7; Ex. Sess. 1921, c. 44, s. 3; C. S. 6612.)

§ 90-9. Examination for license; scope; conditions and prerequisites.—It shall be the duty of the board of medical examiners to examine for license to practice medicine or surgery, or any of the branches thereof, every applicant who complies with the following provisions: He shall, before he is admitted to examination, satisfy the board that he has an academic education equal to the entrance requirements of the University of North Carolina, or furnish a certificate from the superintendent of public instruction of the county that he has passed an examination upon his literary attainments to meet the requirements of entrance in the regular course of the state university. He shall exhibit a diploma or furnish satisfactory proof of graduation from a medical college in good standing requiring an attendance of not less than four years, and supplying such facilities for clinical and scientific instruction as shall meet the approval of the board; but the requirement of four years attendance at a school shall not apply to those graduating prior to January the first, nineteen hundred.

The examination shall cover the following branches of medical science: anatomy, embryology, histology, physiology, pathology, bacteriology, surgery, pediatrics, medical hygiene, chemistry, pharmacy, materia medica, therapeutics, obstetrics, gynecology, and the practice of medicine.

If on such examination the applicant is found competent, the board shall grant him a license authorizing him to practice medicine or surgery or any of the branches thereof.

Five members of the board shall constitute a quorum, and four of those present shall be agreed as to the qualifications of the applicant. (Rev., s. 4498; 1913, c. 20, ss. 2, 3, 6; 1921, c. 47, s. 1; C. S. 6613.)

§ 90-10. Two examinations, preliminary and final, allowed.—It shall be the duty of the state board of medical examiners to examine any applicant for license to practice medicine on the subjects of anatomy, histology, physiology, bacteriology, embryology, pathology, medical hygiene, and chemistry, upon his furnishing satisfactory evidence from a medical school in good standing, and supplying such facilities for anatomical and laboratory instruction as shall meet with the approval of the board, that he has completed the course of study in the school upon the subjects mentioned. The board shall set to the credit of such applicant upon its record books the grade made by him upon the examination, which shall stand to the credit of such applicant; and when he has subsequently completed the full course in medicine and presents a diploma of graduation from a medical college in good standing, requiring a four years course of study of medicine for graduation, and when he has completed the examination upon the further branches of medicine, to wit, pharmacy, materia medica, therapeutics, obstetrics, gynecology, pediatrics, practice of medicine and surgery, he shall have accounted to his credit the grade made upon the former examination, and if then upon such completed examination he be found competent, said board shall grant him a license to practice medicine and surgery, and any of the branches thereof. (1921, c. 47, s. 2; Ex. Sess. 1921, c. 44, s. 4; C. S. 6614.)

§ 90-11. Qualification of applicant for license.—Every person making application for a license to practice medicine or surgery in the state shall be not less than twenty-one years of age, and of good moral character, before any license can be granted by the board of medical examiners: Provided, that the age requirement shall not apply to students tak-

ing the examinations of the first two years in medicine. (1921, c. 47, s. 3; Ex. Sess. 1921, c. 44, s. 5; C. S. 6615.)

§ 90-12. Limited license.—The board may, whenever in its opinion the conditions of the locality where the applicant resides are such as to render it advisable, make such modifications of the requirements of the preceding sections, both as to application for examination and examination for license, as in its judgment the interests of the people living in that locality may demand, and may issue to such applicant a special license, to be entitled a "Limited License," authorizing the holder thereof to practice medicine and surgery within the limits only of the districts specifically described therein. The holder of the limited license practicing medicine or surgery beyond the boundaries of the district as laid down in said license shall be guilty of a misdemeanor, and upon conviction shall be fined not less than twenty-five dollars nor more than fifty dollars for each and every offense; and the board is empowered to revoke such limited license, in its discretion, after due notice. The clerk of the superior court, in registering the holder of a limited license, shall copy upon the certificate of registration and upon his record the description of the district given in the license. (1909, c. 218, s. 1; C. S. 6616.)

§ 90-13. When license without examination allowed.—The board of medical examiners shall in their discretion issue a license to any applicant to practice medicine and surgery in this state without examination if said applicant exhibits a diploma or satisfactory proof of graduation from a medical college in good standing, requiring an attendance of not less than four years and a license issued to him to practice medicine and surgery by the board of medical examiners of another state. (1907, c. 890, 1913, c. 20, s. 3; C. S. 6617.)

§ 90-14. Board may rescind license.—The board shall have the power to revoke and rescind any license granted by it, when, after due notice and hearing, it shall find that any physician licensed by it has been guilty of grossly immoral conduct, or of producing or attempting to produce a criminal abortion, or, by false and fraudulent representations, has obtained or attempted to obtain, practice in his profession, or is habitually addicted to the use of morphine, cocaine or other narcotic drugs, or is habitually addicted to the use of marijuana, barbiturates, demerol or any other habit forming drug or derivative of such drug, or has by false or fraudulent representations of his professional skill obtained, or attempted to obtain, money or anything of value, or has advertised or held himself out under a name other than his own, or has advertised or publicly professed to treat human ailments under a system or school of treatment or practice other than that for which he holds a license, or is guilty of any fraud or deceit by which he was admitted to practice, or has been guilty of any unprofessional or dishonorable conduct unworthy of, and affecting, the practice of his profession, or has been convicted in any court, state or federal, of any felony or other criminal offense involving moral turpitude, or has been adjudicated a mental incompetent or whose mental condition renders him unable safely to practice medicine. And, for any of the above reasons, the said board of medical examiners may refuse to issue a license to an applicant. The findings and actions of the Board of Medical Examiners in revoking or rescinding and refusing to issue licenses under this Section shall be subject to review upon appeal to the Superior Court, as hereinafter provided in this Article. The said Board of Medical Examiners may, in its discretion, restore a license so revoked and rescinded, upon due notice being given and hearing had, and satisfactory evidence produced of reformation of the licentiate. (1921, c. 47, s. 4; Ex. Sess. 1921, c. 44, s. 6; 1933, c. 32; C. S. 6618, 1953, c. .)

§ 90-14.1. Judicial review of board's decision denying issuance of a license. — Whenever the Board of Medical Examiners has determined that a person has duly made application to take an examination to be given by the board showing his education, training and other qualifications required by said board, or that a person who has taken and passed an examination given by the board, has failed to satisfy the board of his qualifications to be examined or to be issued a license, for any cause other than failure to pass an examination, the board shall immediately notify such person of its decision, and indicate in what respect the applicant has so failed to satisfy the board. Such applicant shall be given a formal hearing before the board upon request of such applicant filed with or mailed by registered mail to the secretary of the board at Raleigh, N. C., within 10 days after receipt of the board's decision, stating the reasons for such request. The board shall within 20 days of receipt of such request notify such applicant of the time and place of a public hearing, which shall be held within a reasonable time. The burden of satisfying the board of his qualifications for licensure shall be upon the applicant. Following such hearing, the board shall determine whether the applicant is qualified to be examined or is entitled to be licensed as the case may be. Any such decision of the board shall be subject to judicial review upon appeal to the Superior Court of Wake County upon the filing with the board of a written notice of appeal with exceptions taken to the decision of the board within 20 days after service of notice of the board's final decision. Within 30 days after receipt of notice of appeal, the secretary of the board shall certify to the Clerk of the Superior Court of Wake County the record of the case which shall include a copy of the notice of hearing, a transcript of the testimony and evidence received at the hearing, a copy of the decision of the board, and a copy of the notice of appeal and exceptions. Upon appeal the case shall be heard by the judge without a jury, upon the record, except that in cases of alleged omissions or errors in the record, testimony may be taken by the court. The decision of the board shall be upheld unless the substantial rights of the applicant have been prejudiced because the decision of the board is in violation of law, or is not supported by any evidence admissible under this Article, or is arbitrary or capricious. Each party to the review proceeding may appeal to the Supreme Court as hereinafter provided in Section 90-14.11.

§ 90-14.2. Hearing before revocation or suspension of a license.—Before the board shall revoke or rescind any license granted by it to any physician, it will give to the physician a written notice indicating the general nature of the charges, accusation, or complaints preferred against him and stating that the licensee will be given an opportunity to be heard concerning such charges or complaints at a time and place stated in such notice, or to be thereafter fixed by the board, and shall hold a public hearing not less than 30 days from the date of the service of such notice upon such licensee, at which he may appear personally or through counsel, may cross-examine witnesses and present evidence in his own behalf. A physician who is mentally incompetent shall be represented at such hearing and shall be served with notice as herein provided by and through a guardian ad litem appointed by the clerk of the court of the county in which the physician has his residence. Such licensee or physician may, if he desires, file written answers to the charges or complaints preferred against him within

30 days after the service of such notice, which answer shall become a part of the record but shall not constitute evidence in the case.

§ 90-14.3. **Service of notices.**—Any notice required by this Chapter may be served either personally or by an officer authorized by law to serve process, or by registered mail, return receipt requested, directed to the licensee or applicant at his last known address as shown by the records of the board. If notice is served personally, it shall be deemed to have been served at the time when the officer delivers the notice to the person addressed. Where notice is served by registered mail, it shall be deemed to have been served on the date borne by the return receipt showing delivery of the notice to the addressee or refusal of the addressee to accept the notice.

§ 90-14.4. **Place of hearings for revocation or suspension of license.**—Upon written request of the accused physician, given to the secretary of the board 20 days after service of the charges or complaints against him, a hearing for the purpose of determining revocation or suspension of his license shall be conducted in the county in which such physician maintains his residence, or at the election of the board, in any county in which the act or acts complained of occurred. In the absence of such request, the hearing shall be held at a place designated by the board, or as agreed upon by the physician and the board.

§ 90-14.5. **Use of trial examiner or depositions.**—Where the licensee requests that the hearing herein provided for be held by the board in a county other than the county designated for the holding of the meeting of the board at which the matter is to be heard, the board may designate in writing one or more of its members to conduct the hearing as a trial examiner or trial committee, to take evidence and report a written transcript thereof to the board at a meeting where a majority of the members are present and participating in the decision. Evidence and testimony may also be presented at such hearings and to the board in the form of depositions taken before any person designated in writing by the board for such purpose or before any person authorized to administer oaths, in accordance with the procedure for the taking of depositions in civil actions in the Superior Court.

§ 90-14.6. **Evidence admissible.** — In proceedings held pursuant to this Article the board shall admit and hear evidence in the same manner and form as prescribed by law for civil actions. A complete record of such evidence shall be made, together with the other proceedings incident to such hearing.

§ 90-14.7. **Procedure where person fails to request or appear for hearing.**—If a person who has requested a hearing does not appear, and no continuance has been granted, the board or its trial examiner or committee may hear the evidence of such witnesses as may have appeared, and the board may proceed to consider the matter and dispose of it on the basis of the evidence before it. For good cause, the board may reopen any case for further hearing.

§ 90-14.8. **Appeal from board's decision revoking or suspending a license.**—A physician whose license is revoked or suspended by the board may obtain a review of the decision of the board in the Superior Court of Wake County or in the Superior Court in the county in which the hearing was held or upon agreement of the parties to the appeal in any other Superior Court of the State, upon filing with the secretary of the board a written notice of appeal within 20 days after the date of the service of the decision of the board, stating all exceptions taken to the decision of the board and indicating the court in which the appeal is to be heard.

Within 30 days after the receipt of a notice of appeal as herein provided, either by an applicant or a licensee, the board shall prepare, certify and file with the Clerk of the Superior Court in the county to which the appeal is directed the record of the case comprising a copy of the charges, notice of hearing, transcript of testimony, and copies of documents or other written evidence produced at the hearing, decision of the board, and notice of appeal containing exceptions to the decision of the board.

§ 90-14.9. **Appeal bond; stay of board order.**—The person seeking the review shall file with the clerk of the reviewing court a copy of the notice of appeal and an appeal bond of $200 at the same time the notice of appeal is filed with the board. At any time before or during the review proceeding the aggrieved person may apply to the reviewing court for an order staying the operation of the board decision pending the outcome of the review, which the court may grant or deny in its discretion.

§ 90-14.10. **Scope of review.**—Upon the review of the board's decision revoking or suspending a license, the case shall be heard by the judge without a jury, upon the record, except that in cases of alleged omissions or errors in the record, testimony thereon may be taken by the court. The court may affirm the decision of the board or remand the case for further proceedings; or it may reverse or modify the decision if the substantial rights of the acused physician have been prejudiced because the findings or decisions of the board are in violation of substantive or procedural law, or are not supported by competent, material, and substantial evidence admissible under this Article, or are arbitrary or capricious. At any time after the notice of appeal has been filed, the court may remand the case to the board for the hearing of any additional evidence which is material and is not cumulative, and which could not reasonably have been presented at the hearing before the board.

§ 90-14.11. **Appeal to Supreme Court; appeal bond.**—Any party to the review proceeding, including the board, may appeal to the Supreme Court from the decision of the Superior Court under rules of procedure applicable in other civil cases. No appeal bond shall be required of the board. The appealing party may apply to the Superior Court for a stay of that court's decision or a stay of the board's decision, whichever shall be appropriate, pending the outcome of the appeal to the Supreme Court.

§ 90-14.12. **Injunctions.**—The board may appear in its own name in the Superior Courts in an action for injunctive relief to prevent violation of this Article and the Superior Courts shall have power to grant such injunctions regardless of whether criminal prosecution has been or may be instituted as a result of such violations. (Sections 90-14.1 through 90-14.12 were added by 1953 Session.)

§ 90-15. **License fee; salaries, fees and expenses of board.**—Each applicant for a license by examination shall pay to the treasurer of the Board of Medical Examiners of the State of North Carolina a fee which shall be prescribed by said board in an amount not exceeding the sum of fifty dollars ($50.00) before being admitted to the examination: Provided, however, that in the case of applicants taking the examination in two halves, as provided in Section 90-10, one-half of the prescribed fee shall be paid by the applicant for each of the two half examinations. Whenever any license is granted without examination, as authorized in Section 90-13, the applicant shall pay to the treasurer of the board a fee in an amount to be prescribed by the board not in excess of one hundred dollars ($100.00). Whenever a limited license is granted as provided in Section 90-12, the applicant shall pay to the treasurer

of the board a fee of fifty dollars ($50.00), except where a limited license to practice within the confines of a hospital for the purpose of education or training, the applicant shall pay a fee of ten dollars ($10.00). A fee of ten dollars ($10.00) shall be paid for the issuance of a duplicate license. All fees shall be paid in advance to the treasurer of the Board of Medical Examiners of the State of North Carolina, to be held by him as a fund for the use of said board. The compensation and expenses of the members and officers of the said board and all expenses proper and necessary in the opinion of the board to the discharge of its duties under and to enforce the laws regulating the practice of medicine or surgery shall be paid out of said fund, upon the warrant of the president and secretary of said board. The salaries and fees of the officers and members of said board shall be fixed by the board but shall not exceed ten dollars ($10.00) per day per member for time spent in the performance and discharge of his duties as a member of said board, and reimbursement for travel and other necessary expenses incurred in the performance of his duties as a member of said board. Any unexpended sum or sums of money remaining in the treasury of said board at the expiration of the terms of office of the members thereof shall be paid over to their successors in office. (Rev. s. 4501; Code s. 3130 1858-9, c. 258, s. 13; 1913, c. 20, ss. 4-5; 1921, c. 47 s. 5; Ex. Sess. 1921, c. 44, s. 7; C. S. 6619; amended, Session 1953.)

§ 90-16. Board to keep record; publication of names of licentiates; transcript as evidence. — The board of examiners shall keep a regular record of its proceedings in a book kept for that purpose, together with the names of the members of the board present, the names of the applicants for license, and other information as to its actions. The board of examiners shall cause to be entered in a separate book the name of each applicant to whom a license is issued to practice medicine or surgery, along with any information pertinent to such issuance. The board of examiners shall publish the names of those licensed in three daily newspapers published in the state of North Carolina, within thirty days after granting the same. A transcript of any such entry in the record books, or a certificate that there is not entered therein the name and proficiency or date of granting such license of a person charged with the violation of the provisions of this article, certified under the hand of the secretary and the seals of the board of medical examiners of the state of North Carolina, shall be admitted as evidence in any court of this state when it is otherwise competent. (Rev., s. 4500; Code, s. 3129; 1858-9, c. 258, s. 12; 1921, c. 47, s. 6; C. S. 6620.)

§ 90-17. Blanks furnished clerk.—It shall be the duty of the medical society of the state of North Carolina to prescribe proper form of certificates required by this article and all such blanks and forms as the clerk may need to enable him to perform his duties under this article. (Rev., s. 4505; 1889, c. 181, s. 7; 1899, c. 93, s. 4; C. S. 6621.)

§ 90-18. Practicing without license; practicing defined; penalties.—No person shall practice medicine or surgery, or any of the branches thereof, nor in any case prescribe for the cure of diseases unless he shall have been first licensed and registered so to do in the manner provided in this article, and if any person shall practice medicine or surgery without being duly licensed and registered, as provided in this article, he shall not be allowed to maintain any action to collect any fee for such services. The person so practicing without license shall be guilty of a misdemeanor, and upon conviction thereof shall be fined not less than fifty dollars ($50) nor more than one hundred ($100), or imprisoned at the discretion of the court for each and every offense.

Any person shall be regarded as practicing medicine or surgery within the meaning of this article who shall diagnose or attempt to diagnose, treat or attempt to treat, operate or attempt to operate on, or prescribe for or administer to, or profess to treat any human ailment, physical or mental, or any physical injury to or deformity of another person: Provided, that the following cases shall not come within the definition above recited:

1. The administration of domestic or family remedies in cases of emergency.

2. The practice of dentistry by any legally licensed dentist engaged in the practice of dentistry and dental surgery.

3. The practice of pharmacy by any legally licensed pharmacist engaged in the practice of pharmacy.

4. The practice of medicine and surgery by any surgeon or physician of the United States army, navy, or public health service in the discharge of his official duties.

5. The treatment of the sick or suffering by mental or spiritual means without the use of any drugs or other material means.

6. The practice of optometry by any legally licensed optometrist engaged in the practice of optometry.

7. The practice of midwifery by any woman who pursues the vocation of midwife.

8. The practice of chiropody by any legally licensed chiropodist when engaged in the practice of chiropody, and without the use of any drug.

9. The practice of osteopathy by any legally licensed osteopath when engaged in the practice of osteopathy as defined by law, and especially § 90-129.

10. The practice of chiropractic by any legally licensed chiropractor when engaged in the manual adjustment of the twenty-four spinal vertebrae of the human body and without the use of drugs.

11. The practice of medicine or surgery by any reputable physician or surgeon in a neighboring state coming into this state for consultation with a resident registered physician. This proviso shall not apply to physicians resident in a neighboring state and regularly practicing in this state.

12. Physicians who have a diploma from a regular medical college or were practicing medicine and surgery in this state prior to the seventh day of March, one thousand eight hundred and eighty-five, and who are properly registered as required by law.

13. Any person practicing Radiology as hereinafter defined shall be deemed to be engaged in the practice of medicine within the meaning of this article. "Radiology" shall be defined as, that method of medical practice in which demonstration and examination of the normal and abnormal structures, parts or functions of the human body are made by use of x-rays. Any person shall be regarded as engaged in the practice of Radiology who makes or offers to make for a consideration, a demonstration or examination of a human being or a part or parts of a human body by means of fluoroscopic exhibition or by the shadow imagery registered with photographic materials and the use of x-rays; or holds himself out to diagnose or able to make or makes any interpretation or explanation by word of mouth, writing or otherwise of the meaning of such fluoroscopic or registered shadow imagery of any part of the human body by use of x-rays; or who treats any disease or condition of the human body by the application of x-rays or radium. Nothing in this subsection shall prevent the practice of Radiology by any person licensed under the provisions of Articles 2, 5, 6, and 11 of chapter 110. (Rev., ss. 3645, 4502;

Code, s. 3122; 1858-9, c. 258, s. 2; 1885, c. 117, s. 2; 1885, c. 261; 1889, c. 181, ss. 1, 2; 1921, c. 47, s. 7; Ex. Sess. 1921, c. 44, s. 8; 1941, c. 163; C. S. 6622.)

§ 90-19. Practicing without registration; penalties.—Any person desiring to engage in the practice of medicine or surgery shall personally appear before the clerk of the superior court of the county in which he resides or practices, for registration as a physician or surgeon. The person so applying shall produce and exhibit before the clerk of the superior court a license obtained from the board of medical examiners of the state. The clerk shall thereupon register the date of registration, with the name and residence of such applicant, in a book to be kept for this purpose in his office marked "Register of Physicians and Surgeons," and shall issue to him a certificate of registration under the seal of the superior court of the county upon the form furnished him by the medical society of North Carolina, for which the clerk shall be entitled to collect from said applicant a fee of twenty-five cents. The person obtaining such certificate shall be entitled to practice medicine or surgery, or both, in the county where the same was obtained, and in any other county in this state; but if he shall remove his residence to another county he shall exhibit said certificate to the clerk of such other county and be registered, which registration shall be made by said clerk without fee or charge.

Any person who practices or attempts to practice medicine or surgery in this state without first having registered and obtained the certificate required in this section, shall be guilty of a misdemeanor, and upon conviction thereof shall be fined not less than twenty-five dollars nor more than one hundred dollars, or be imprisoned at the discretion of the court, for each and every offense: Provided, this section shall not apply to women pursuing the vocation of midwife, nor to reputable physicians or surgeons resident in a neighboring state coming into this state for consultation with a registered physician of this state. (Rev., ss. 3646, 4504; 1889, c. 181, ss. 5; 1891, c. 420; Ex. Sess. 1921, c. 44, s. 9; C. S. 6623.)

§ 90-20. Clerk punishable for illegally registering physician.—If any clerk of the superior court shall register, or issue a certificate to, any person practicing medicine or surgery in any other manner than that prescribed by law, he shall be guilty of a misdemeanor, and upon conviction thereof shall be fined not less than two hundred dollars and shall be removed from office. (Rev., s. 3647; 1889, c. 181, s. 6; C. S. 6624.)

§ 90-21. Certain offenses prosecuted in superior court: duties of attorney-general.—In case of the violation of the criminal provisions of §§ 90-18 to 90-20, the attorney-general of the state of North Carolina, upon complaint of the board of medical examiners of the state of North Carolina, shall investigate the charges preferred, and if in his judgment the law has been violated, he shall direct the solicitor of the district in which the offense was committed to institute a criminal action against the offending persons. A solicitor's fee of five dollars shall be allowed and collected in accordance with the provisions of §§ 6-12. The board of medical examiners may also employ, at their own expense, special counsel to assist the attorney-general or the solicitor.

Exclusive original jurisdiction of all criminal actions instituted for the violations of §§ 90-18 to 90-20 shall be in the superior court, the provisions of any special or local act to the contrary notwithstanding. (1915, c. 220, s. 2; C. S. 6625.)

PUBLIC RELATIONS

WHY THE PRIVATE PRACTICE OF MEDICINE FURNISHES THIS COUNTRY WITH THE FINEST MEDICAL CARE

THOMAS RAMON BELLO

REIDSVILLE

We are now standing on a threshold of a half-century of miraculous medical progress. In these 50 years we have taken tremendous strides toward wiping out the diseases of the past century. Many of the infections and deadly diseases suffered during our grandparent's generation — scarlet and typhoid fever, diphtheria, whooping cough, measles —have either practically disappeared or been reduced to minor illnesses. Advances have been made toward the cure of tuberculosis, infantile paralysis, cancer, and heart disease. A decade ago, there was no mention of cortisone, antihistimine, ACTH, vitamin B_{12}, penicillin, or the more recent antibiotics. The life expectancy has been increased by 20 years, and the death rate has been cut almost in half — saving from disease and death almost one million lives a year. Our American mothers have better than 999 out of 1000 chances to pull through childbirth safely—the best record in our history and, on a comparable basis, the best in the world. This enviable record has been built in an atmosphere of freedom and enterprise by some of the world's greatest people—the American doctors and scientists.

Effect of Socialization

Now there are people that would have this progress halted and this system of free medical enterprise ended. They believe that the government should control medicine—thereby furnishing greater medical care for more people at a greatly reduced cost to the individual. Could this happen in our country and be a benefit to our people, our working man?

Let us review the effect that socialization has had on medicine in England. In our country the wage earner works two days out of six to pay all his various taxes. In England, the worker has to add a third day—making him work one-half his time to pay the government for a medical service that

Prize-winning essay in the high school essay contest sponsored by the Public Relations Committee.

he is not even sure he will get. Each worker has the cost of medical aid subtracted regularly from his pay roll for unused aspirin, bandages, and drugs when he really needs the money for food and clothing. Whenever he does become ill, he will be one of 50 patients, half of whom are bent on getting service for their money, whom a physician must see in an *hour!* The physician merely has time to ask few questions and to fill out one of 18 different forms to go to the government. A patient can receive only mediocre treatment from overworked doctors. He becomes a number in a written form instead of an individual patient.

If a man earns between two and three thousand dollars a year and pays insurance for a period of 40 working years to take care of his illness, would the government unconditionally guarantee a bed and a physician at the critical moment when his wife is in labor? At the operating table, could the government provide a surgeon in whose hands a man could trustingly lay his life? It has been proven many times that the government will not come through with its side of the "bargain," even when human life is at stake. Doctors also would have to look out for themselves. Since the doctors would have an insured income from the government, they would lack initiative; and, therefore, the quality of medicine would drastically fall. Surely our free practice of medicine furnishes the best medical care to the individual and wage earner.

Progress Under a Free System

In the United States, we believe that every man may develop his own will and initiative that the end product may be beneficial to humanity, his country, and himself. It is under this sound belief and doctrine that our policy of free business and enterprise has developed. By this doctrine of freedom and individual initiative, our medical profession has developed unto utmost heights. In this field, the government has been a tremendous *aid* to the people Under the Hill-Burton Act passed by Congress in 1946, 1,900 hospital projects have been approved, of which 1,010 have been completed with federal aid in every state and territory. More than one half of these new hospitals have been built in towns of less than 5,000 inhabitants. The projects will add 90,000 hospital beds, 360 public health centers, and 15 state laboratories in the nation's total. The initiative in efforts to get Hill-Burton funds for a hospital is taken by the local community. After completion the hospital belongs to the community. The federal government makes its contribution to the community, *then gets out!* Yes, federal control is one matter, federal aid another!

Let us look at a recent practical application of medical advancement under a free system. In Bat Cave, North Carolina, Dr. George F. Bond had wearied of a saddle bag practitioner's existence and a lack of working facilities. Appearing before the people, he persuaded them to build a 16-bed clinic. Now he can treat effectively 90 per cent more patients than he did before. Under government control this community project would have not been possible at all. It is this free initiative that raises the quality of our medical service.

Several states now have programs to get more physicians to move to the rural areas and practice. One state of note in this program is Colorado, which has already placed a doctor in almost every community that can support one; 5 of 17 planned new hospitals are now in use. During the past three years, the Iowa State Medical Society has placed 159 physicians in previously doctorless communities. Since the end of World War II, almost completely rural South Dakota has gained 138 physicians.

One big problem prevailing both in free and socialized countries is long illness that can become a catastrophe to any family except those in the highest income brackets. Again government control is at a disadvantage in comparison to local initiative. The California Physicians Service has introduced a catastrophe insurance, giving full medical care up to $5,000 for two years for infantile paralysis, cancer, and 21 other diseases. The cost per family is $1.95 per month. No government offering socialized medical care or insurance can beat that. Our own voluntary medical insurance covers a patient through 70 days of hospitalization in one year, as compared to only 60 days per year offered by government medical controls.

Governed by their own free will and initiative, American doctors are constantly striving to make the inhabitants of the United States stronger and healthier than any other people in the world. In fact, Swedes actually live longer in Minnesota than they do in

Sweden. Regardless of the fine medical service that we enjoy, we pay no more in proportion for it than did our parents 20 years ago.

Backing our doctors with the best drugs and medicine are some of the world's best scientists and chemists. Painstakingly these scientists have tracked down some of the world's most dreaded diseases and have all but wiped them out with chemicals. Not long ago pneumonia was the third greatest killer; already deaths from it have dropped 70 per cent—most cases can be cured in just a few days with penicillin, aureomycin, and other antibiotics. A country doctor, aided by the new wonder drugs, has more power to save life in a case of perforating appendicitis than the greatest specialists had a generation ago; yearly deaths have decreased from 17,000 to 5,000. Yet at this very moment, we are suddenly threatened with an end to our voluntary fight against death and pain.

Conclusion

Unless we continue to improve constantly, we will deteriorate. The federal government can help without controlling. A law to eliminate illness is as useless as a law to eliminate war. At this moment we stand before the greatest medical decade in history, promising the control of cancer and the major part of the dreaded diseases of the heart and arteries. We can hope for this progress as long as our doctors, health services, and pharmaceutical companies are left free to continue what they have brilliantly begun. As Mr. Dave Beck said in an address delivered before the one hundredth convention of the A.M.A.:

> "The medical profession has grown to its present high estate under our system of democratic freedoms. The opportunity to meet the challenge of disease and death has been taken by the doctors, using the weapons of science, truth, and skill—all welded within the framework of a free society. That freedom must be preserved. For in that freedom, medicine has grown and will continue to grow if there is no interference with individual initiative. The progress of American medicine must not be blocked in the impediments of an uncertain and clumsy bureaucracy. We have been born as a great people and we will continue to thrive as a great people only under a system of free enterprise—free for the business man, free for labor, free for the doctors, and free for all of our people."

BULLETIN BOARD

COMING MEETINGS

Duke Medical Symposium on Psychiatry for the Non-Psychiatrist—Duke University, December 1-2.

American College of Chest Physicians, Sixth Annual Postgraduate Course—New York City, November 2-6.

National Society for Crippled Children and Adults, 1953 Annual Convention—Chicago, Illinois, November 12-14.

DUKE UNIVERSITY SCHOOL OF MEDICINE SIXTEENTH ANNUAL SYMPOSIUM

The Duke University School of Medicine again cordially invites physicians of the Carolinas, Virginia, and Tennessee to attend a medical symposium on Tuesday and Wednesday, December 1 and 2, 1953, on the Duke University campus. The subject chosen for this year is "Psychiatry For The Non-Psychiatrist." It is intended to renew the acquaintance of the non-psychiatric physician, be he general practitioner or specialist, with the psychiatric aspects of diagnosis and treatment.

The program has three sections. In the first section everyday problems of childhood, of adolescence, and of advanced age will be discussed. The second section is given over to psychosomatics in internal medicine and in obstetrics and gynecology, and to home care, before and after hospitalization, of patients with psychiatric illnesses. The third section will include such topics as "Front Line Psychiatry in Modern War," "Psychiatry and the Practice of Industrial Medicine," and "Planning For Rural Psychiatry." After each lecture there will be ample opportunity for questions and for discussion from the floor.

On Tuesday night there will be a panel on the counseling of relatives concerning psychiatric illness (hospitalization, treatment, prognosis, commitment procedures, and after care). It is hoped that questions on these topics will originate from the audience.

The following speakers have agreed to appear on the program: Spafford Ackerly, M.D., professor of psychiatry, University of Louisville School of Medicine, Louisville, Kentucky; L. H. Bartemeier, M.D., associate professor of psychiatry, Wayne University School of Medicine, and visiting professor of psychiatry, University of Michigan, Detroit, Michigan; F. J. Curran, M.D., professor of neurology and psychiatry, University of Virginia Medical School, and director of the Children's Service Center, Charlottesville, Virginia; A. J. Glass, Colonel MC, USA, chief of neuropsychiatric services, Brooke Army Medical Center, Fort Sam Houston, Texas; R. W. Howell, M.D., professor of mental health, School of Public Health, University of North Carolina, Chapel Hill; W. S. Kroger, M.D., associate clinical professor of obstetrics and gynecology, Chicago Medical School, Chicago, Illinois; W. T. Lhamon, M.D., associate professor of psychiatry, University of Pennsylvania School of Medicine, Philadelphia; Theodore Lidz, M.D., professor of psychiatry, Yale University School of Medicine, New Haven, Connecticut; R. D. Rabinovitch, M.D., chief of Children's Service, Neuropsychiatric Institute, University of Michigan, Ann Arbor, Michigan.

Rural Health Conference

The Sixth Annual Rural Health Conference, sponsored by the Medical Society of North Carolina, was held Wednesday, October 21, at the North Carolina State College Textile Building in Raleigh. Co-sponsors for this year's conference included the North Carolina State Health Council, the North Carolina Grange, the North Carolina Farm Bureau Federation, the Health Committee of the North Carolina Congress of Parents and Teachers, and the Federation of Home Demonstration Clubs along with other agricultural and health organizations.

Commissioner of Agriculture L. Y. Ballentine presided over the morning session. Keynote speaker of the one-day session on the theme "Dividends of Health" was Mrs. Charles W. Sewell of Otterbein, Indiana, former Administrative Director of the American Farm Bureau Associated Women, and co-founder of the rural health movement. Another feature of the program was a dutch family style picnic and a panel discussion on everyday health problems.

A Progress Parade—reports from selected communities on how they determined and solved their health problems—was featured again this year, including a section accenting accomplishments by the youth of North Carolina in the field of health.

Summation of the day's program was made by R. C. Francis of Waynesville. Also on the program was I. G. Greer, executive director, North Carolina Business Foundation, who discussed "Health Dividends for Youth."

North Carolina Public Health Association

Dr. C. C. Applewhite, Director of the State Board of Health's Division of Local Health Administration, was signally honored by the North Carolina Public Health Association at its forty-second annual meeting at Nags Head, when he received the 1953 Carl V. Reynolds Award for distinguished service in the field of public health. The Award was announced at the annual banquet.

North Carolina Society for Crippled Children and Adults

The eighteenth annual meeting of the North Carolina Society for Crippled Children and Adults (the Easter Seal Society) was held in Rocky Mount, October 16 and 17. On the program were speakers from the fields of medicine, education, and rehabilitation.

North Carolina Surgical Association

The fifth annual meeting of the North Carolina Surgical Association was held at Roaring Gap, September 19. The program consisted of a paper entitled "The Surgical Treatment of Valvular Heart Disease," by Dr. William Sealy of Duke Hospital, Durham, followed by a symposium on "Intestinal Obstruction," with Dr. George Wood of High Point as moderator and the following panel of speakers: Dr. William Farmer of Fayetteville, Dr. Addison Brenizer of Charlotte, Dr. J. B. Anderson of Asheville, and Dr. Hubert Patterson of the North Carolina Memorial Hospital, Chapel Hill.

The following officers were elected for 1953-1954: president, Dr. F. M. Simmons Patterson, New Bern; president-elect, Dr. Felda Hightower, Winston-Salem; vice president, Dr. Edward Phifer, Morganton; Secretary-Treasurer, Dr. Alexander Webb, Jr. of Raleigh.

The next meeting of this association will be held in April, 1954, at The Homestead, Hot Springs, Virginia.

Sixth District Medical Society

The Sixth District Medical Society met at State Hospital, Butner, on Wednesday, October 14. The program extended through the afternoon and early evening.

Edgecombe-Nash Medical Society

The Edgecombe-Nash Medical Society held its monthly meeting in Rocky Mount on September 9.

Dr. Boone Grant, in charge of the scientific program, presented Dr. Hans Lowenbach of the Department of Psychiatry, Duke University School of Medicine, whose subject was "Electroencephalograms: Indications and Interpretations."

News Notes

Dr. Robert W. Williams has announced the opening of offices for the practice of general and thoracic surgery in association with Dr. Lockert B. Mason at 1007 Murchison Building, Wilmington.

* * *

Dr. D. Russell Perry, Jr., has opened offices at 207 South Hawthorne Road, Winston-Salem. His practice will be limited to infants and children.

* * *

Dr. Donald H. Lomax has announced the opening of his office for general practice at 707 Barker Street, Salisbury.

National Society for Crippled Children and Adults

Dr. Edward J. McCormick of Toledo, Ohio, president of the American Medical Association, heads a list of distinguished authorities scheduled to speak on the care, treatment, and training of crippled children at the 1953 annual convention of the National Society for Crippled Children and Adults, November 12-14 at Chicago's Palmer House.

Another prominent personality will be Dr. Frances R. Horwich, Chicago, leading child development authority who, as "Miss Frances," has made the "Ding Dong School" a nationally famous program on television. Dr. Horwich will be principal speaker at the annual parents' institute.

Dr. Henry H. Kessler, medical director of the Kessler Institute for Rehabilitation, West Orange, N. J., and counselor in rehabilitation to the National Society, will head a panel on rehabilitation at the convention.

In addition to other outstanding speakers, the convention program will include general sessions, seminars, exhibits, demonstrations, workshops, social events, and other features packed with information and inspiration for rehabilitation workers, volunteers, and parents of crippled children.

Dr. William T. Sanger, president of the National Society, will preside at this year's convention marking the Society's thirtieth annual meeting and thirty-second year of service to the nation's handicapped.

The National Foundation for Infantile Paralysis

Dr. Henry W. Kumm of Chocorua, New Hampshire, and New York City, has been appointed director of research of the National Foundation for Infantile Paralysis, it was announced recently by Basil O'Connor, president.

Dr. Kumm, who had spent 23 years on the staff of the Rockefeller Foundation for Medical Research before joining the National Foundation in July, 1951, replaces Dr. Harry M. Weaver of Bedford Village, New York, who has resigned.

American Society for the Study of Sterility

The American Society for the Study of Sterility announces the opening of the 1954 contest for the most outstanding contribution to the subject of infertility and sterility. The winner will receive a cash award of $1,000, and the essay will appear on the program of the 1954 meeting of the Society. Essays submitted in this competition must be received not later than March 1, 1954. For full particulars concerning requirements of this competition, address The American Society for the Study of Sterility, c/o Dr. Herbert H. Thomas, Secretary, 920 South 19th Street, Birmingham, Alabama.

The author should append on a separate sheet of paper a short biographical sketch of himself and include a photograph to be used in the necessary publicity should he be the winner of the award.

American Public Health Association

A forum on world health and the American people is scheduled for the opening day of the American Public Health Association's eighty-first meeting at the Hotels Statler and New Yorker, November 9 to 13.

The forum will be sponsored by the National Citizens Committee for the World Health Organization, one of 40 related groups meeting concurrently with the American Public Health Association, professional society of more than 12,000 public health workers in the United States, Mexico, Canada and Cuba. It will help set the stage for scientific sessions on latest developments aimed at preventing disease and promoting personal and public health. More than 5,000 delegates are expected to attend.

(BULLETIN BOARD CONTINUED ON PAGE 540)

Classified Advertisements

WANTED — An Obstetrician-Gynecologist who desires an association to complete required training under preceptorship plan. Reply to 27-JC, P. O. Box 790, Raleigh, North Carolina.

FOR SALE: Professional X-Ray unit. Upright model for Fluoroscopy. Four years old, good condition. Attractive price. Reply to Dr. J. O. Redding, 147 McArthur Street, Asheboro, North Carolina.

INTERNIST wishes part time employment and office space in Charlotte. Reply to O-RJ, P. O. Box 790, Raleigh, North Carolina.

An opening in city of twenty-five to thirty thousand population for young eye, ear, nose and throat physician. Reply to 79-HCW, P. O. Box 790, Raleigh, North Carolina.

BOOK REVIEWS

On Burns. Compiled and edited by Nathan A. Womack, M.D. Participants: Dr. William Altemeier, Dr. James Barrett Brown, Dr. W. H. J. Butterfield, Dr. Truman Blocker, Dr. Julian Bruner, Dr. Stuart Cullen, Dr. Sam May, Dr. Carl Moyer, Dr. Sidney Ziffren. 178 pages. Price, $5.50. Springfield, Illinois: Charles C Thomas, 1953.

This small volume is a transcription of the principal papers submitted at a symposium on burns in Des Moines, Iowa, in May, 1951, as a part of the National Civil Defense Program. The material has been edited very little and has been correlated by comments by the editor. The principal subjects covered are the treatment of burn shock, the open and closed methods of treatment of acute burns, the use of blood, plasma, and plasma substitutes, the problems of infection, of nutrition, and of late treatment. The style is informal and will appeal principally to those who have already had experience in the care of acute and of chronic burns. The ideas expressed are for the most part not new, but they are put in their proper perspective on the basis of experimental evidence and wide clinical experience. It is an excellent over-all summary of the best current opinion regarding the management of an individual burn case, with suggestions for the mass handling of burns in a disaster area.

Living With a Disability. By Howard A. Rusk, M.D., and Eugene J. Taylor. 207 pages. Price, $3.50. Garden City, New York: The Blakiston Company, Inc., 1953.

Few individuals during the past ten years have studied the scientific aspects of rehabilitation of the physically handicapped person with such single-minded devotion as Dr. Howard Rusk. His contributions to the physical and emotional factors concerned in rehabilitation are well known to everyone interested in this subject.

As director of the Institute of Physical Medicine and Rehabilitation in New York, he has had an unparalleled opportunity to investigate facilities available to the handicapped person for rehabilitation. In this small volume he has collected those instruments which have proved to be of practical usefulness in the rehabilitation of people who have suffered some degree of physical disability, but who are anxious to achieve independence. The articles described have been selected from a very large number of instruments studied, many of which have proved impractical to the handicapped person.

The book is profusely illustrated, most of the photographs of excellent quality. There is a section of Picture Credits which gives ready reference to those interested in obtaining the instruments illus-in the text. The book is largely devoted to the efforts a person may make in becoming independent in his own home, but there is passing reference to the wide range of possibilities available in industrial employment.

The text is brief but well written and will be a source of inspiration to the disabled person. This book will make an excellent gift to any hemiplegic, paraplegic, victim of poliomyelitis or cerebral palsy. It is required reading for all medical personnel interested in physical rehabilitation.

BULLETIN BOARD

(CONTINUED FROM PAGE 539)

News Notes from the American Medical Association

For TV Audiences

Earmarked "for local medical societies only," the American Medical Association offers four top-notch new television shows to be used exclusively by state and county medical societies to spark their public relations programs. These films were designed for airing on public service time over local TV stations . . . they will not be aired on any national TV network.

Each program deals with a different popular medical subject: (1) "Operation Herbert"—a fast-moving humorous play discussing medical care costs, stars well-known actor Jackie Kelk; (2) "A Citizen Participates"—documentary-type story tells about a town that needs a doctor and how one citizen heads a campaign to get one; (3) "What to Do"—series of six five-minute shows with ABC women's commentator Nancy Craig demonstrating how to use a thermometer, apply artificial respiration, stock a medicine chest, and emergency treatment of colds, abdominal pains and headaches; and (4) "Your Doctor"—Louise de Rochemont film now available for first time on TV, describes medical training facilities today and focuses on career of a GP in a rural mountain community.

Prints of these new filmed TV shows may be obtained by writing to the AMA's TV Film Library, 535 North Dearborn Street, Chicago 10, Ill. Be sure to book these shows well in advance of schedule date.

* * *

Plan Now For St. Louis!

Physicians and AMA staff members are completing arrangements for the seventh annual Clinical Session to be held December 1 to 4 in St. Louis. Such topics as chest injuries, compression fractures of the vertebra, chronic pancreatitis, weed poisoning and chronic arthritis—drugs and vaccine therapy, will be presented. Special features include—fracture demonstrations; problems of delivery, manikin demonstrations by leading obstetricians; traffic accidents, an exhibit symposium combining the experiences of physicians, the National Safety Council and police departments, and stressing the responsibility of the physician in the prevention of such accidents, and diabetes, which includes exhibits and question-and-answer conferences in an adjoining room.

The Jefferson Hotel has been selected as the headquarters for House of Delegates sessions and Reference Committee meetings. All other features—scientific exhibit, lectures, motion pictures, color television, technical exhibits—will be presented at the Kiel Auditorium. More than 80 exhibits, with continuous demonstrations and plenty of time for personal consultation, will make up the Scientific Exhibit. The Technical Exhibit will feature approximately 150 displays covering all types of office and medical practice needs.

* * *

New Exhibits on Accidents and Immunization

Leading causes of accidents and how to prevent them . . . various immunizing agents and the diseases they prevent . . . comprise the themes of two new health exhibits now available from the American Medical Association.

* * *

Report on Grievance Committees

Just off the presses is a detailed report on county medical society grievance committees. Prepared by the AMA's Council on Medical Service, this study deals with the organization, functions and operations of 198 mediation committees throughout the country. To make the data of more practical value, the societies have been divided into groups according to size. Copies of the booklet are available from the Council.

* * *

Medicine Strikes at "Big Game"

What medicine is doing to overcome the principal causes of death in the world today is the theme of a new series of radio transcriptions released by the A.M.A. September 15. The 13-program series — "Medicine Fights the Killers"—covers the following subjects: coronary thrombosis, rheumatic heart disease, lung cancer, leukemia, hypertension, accidents, nephritis, premature births, tuberculosis, pneumonia, diabetes, suicide and poliomyelitis. Prepared by the Bureau of Health Education, these transcriptions may be aired over local radio stations under the auspices of state and county medical societies.

* * *

Public Relations Conference in St. Louis

The AMA's sixth annual National Medical Public Relations Conference will be held Monday, November 30—the day before the opening of the Clinical Sessions—at the Jefferson Hotel, St. Louis. The Conference program will be geared primarily for physicians. Members of the House of Delegates, officers of state and county medical societies, officers of the Association and executive secretaries, and PR personnel are cordially invited.

Department of Defense

Redistribution to the Navy and Air Force of approximately 500 volunteer doctors recently commissioned in the Army is being undertaken, the Department of Defense announced recently.

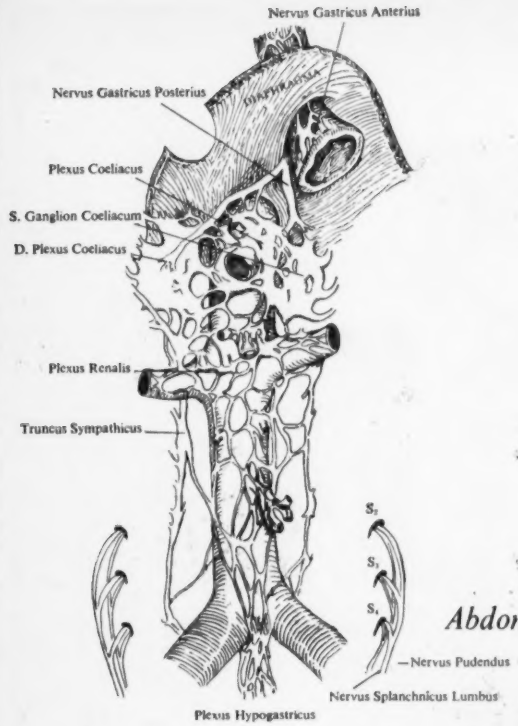
The rather large number of available volunteers is due to the number of physicians who shortly after the Doctor Draft Act was extended for an additional two years, and after the August draft call was announced, decided to volunteer for service now rather than wait until called by their draft boards, Defense Department officials point out.

An additional reason cited by the Department is that many of the doctors now on duty in the Army, who are entitled to early release under the new Doctor Draft Act, are not taking advantage of this opportunity. A sharp decrease in the number of physicians being granted deferments for additional professional training also contributes to the present situation.

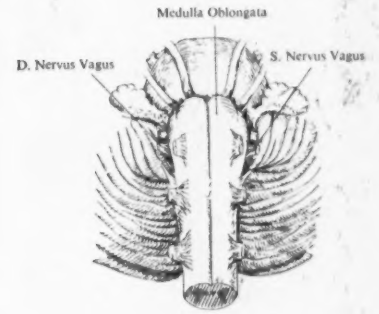
The Navy and Air Force recently have not been accepting volunteers since both of these services temporarily had a number of physicians adequate to meet their needs. Because of scheduled losses, however, these services are now in a position to use some of the Army's volunteers.

Insofar as possible, the redistribution will be accomplished by voluntary interservice transfer; that is, by transferring the commissions of the doctors concerned from the Army to the Navy and Air Force. However, if the number of physicians desiring interservice transfer is insufficient, then it may be necessary to detail some Army doctors to duty with the Navy and Air Force, the Department of Defense points out.

No further draft calls for physicians will be made and no more volunteer medical officers accepted until such time as losses create new vacancies.



Central origin of the vagus nerves (parasympathetic)



Abdominal autonomic plexus (sympathetic)

Control of Gastric Motility and Spasticity in Peptic Ulcer with Banthine®

"The need[1] for suppressing gastric motility and spastic states is . . . fundamental in peptic ulcer therapy. Since the cholinergic nerves are motor and secretory to the stomach and motor to the intestines, agents capable of blocking cholinergic nerve stimulation are frequently used to lessen motor activity and hypermotility."

Banthine[2] "has dual effectiveness; it inhibits acetylcholine liberated at the postganglionic parasympathetic nerve endings and it blocks acetylcholine transmission through autonomic ganglia."

It has been shown[1] to diminish gastric motility and secretion significantly as well as intestinal and colonic motility.

The usual schedule of administration in peptic ulcer is 50 to 100 mg. every six hours, day and night, with subsequent adjustment to the patient's needs and tolerance. After the ulcer is healed, maintenance therapy, approximately half of the therapeutic dosage, should be continued for reasonable assurance of nonrecurrence.

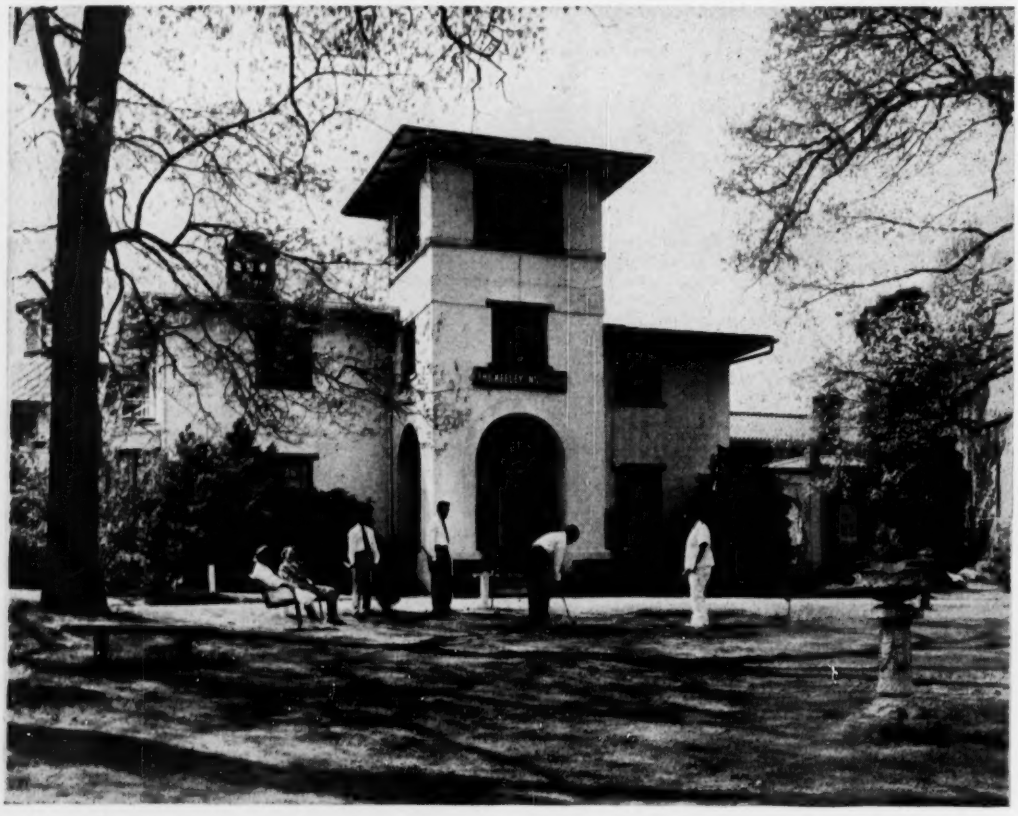
Banthine® (brand of methantheline bromide) is supplied in: Banthine ampuls, 50 mg.—Banthine tablets, 50 mg.

It is accepted by the Council on Pharmacy and Chemistry of the American Medical Association.

1. Zupko, A. G.: Pharmacology and the General Practitioner, GP 7:55 (March) 1953.

2. McHardy, G. G., and Others: Clinical Evaluation of Methantheline (Banthine) Bromide in Gastroenterology, J.A.M.A. *147*:1620 (Dec. 22) 1951.

SEARLE Research in the Service of Medicine



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Oral Surgery:
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Plastic Surgery:
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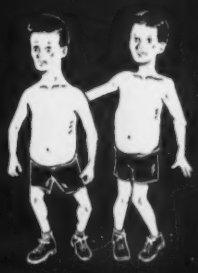
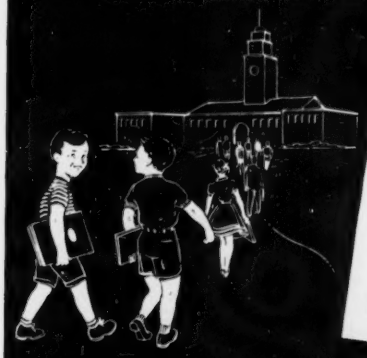
"... in a word, deficiency of fat in the food is one of the chief factors in the production of rickets." (1)



TODAY...



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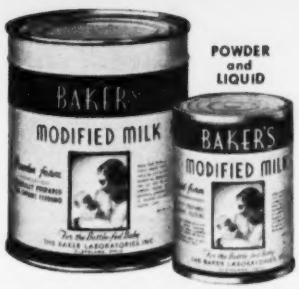

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Vitamin A	2500 U.S.P. Units	Thiamine	0.6 milligrams
Vitamin D	800 U.S.P. Units	Niacin	5.0 milligrams
Vitamin C	None	Riboflavin	1.0 milligrams

A source of vitamin C should be prescribed by the physician.

(1) Cheadle—*Artificial Feeding and Food Disorders of Infants*. Sixth Edition (1906)

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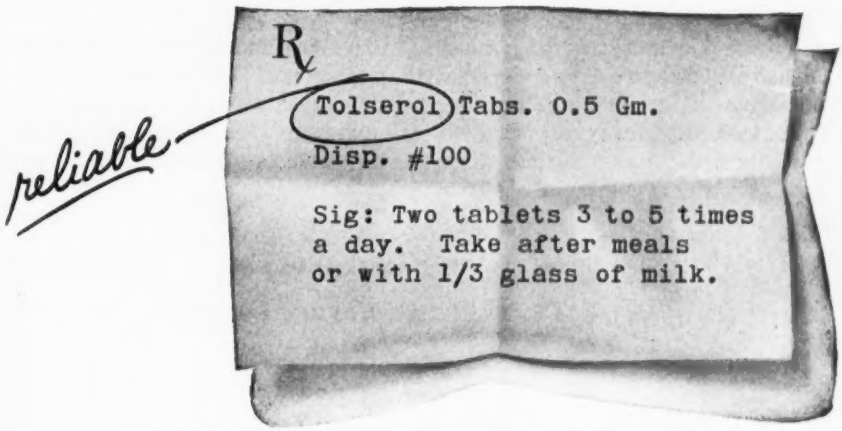

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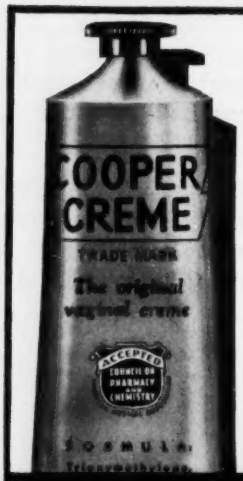

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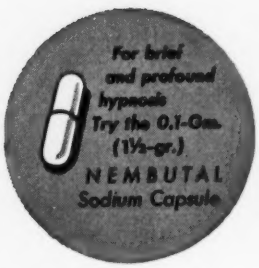

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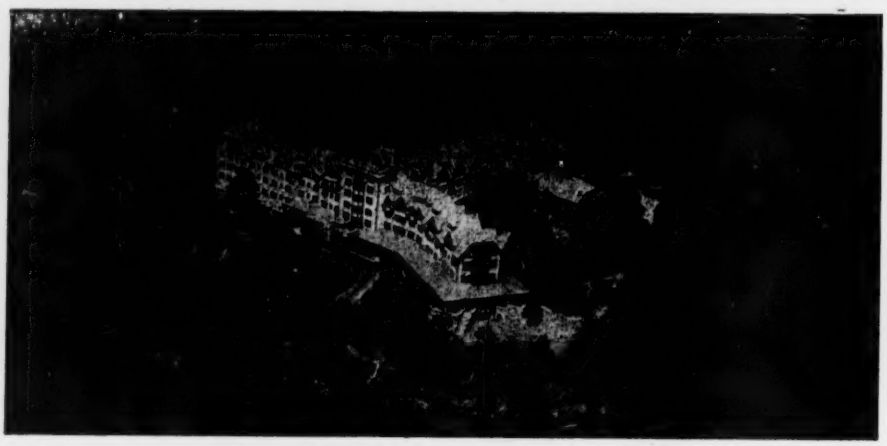

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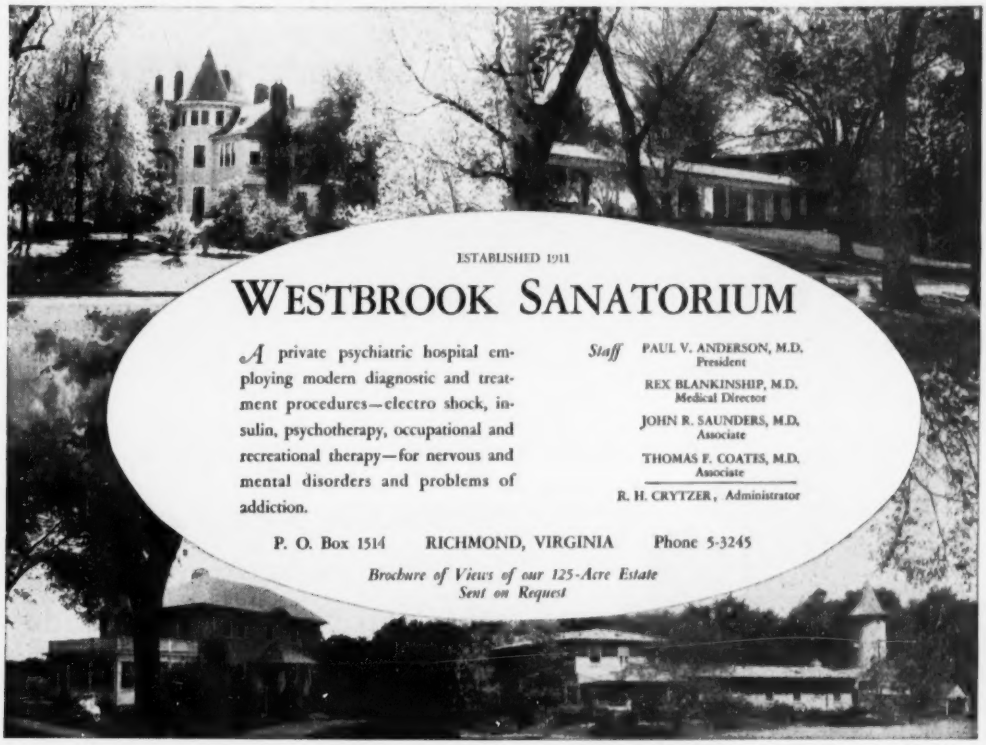
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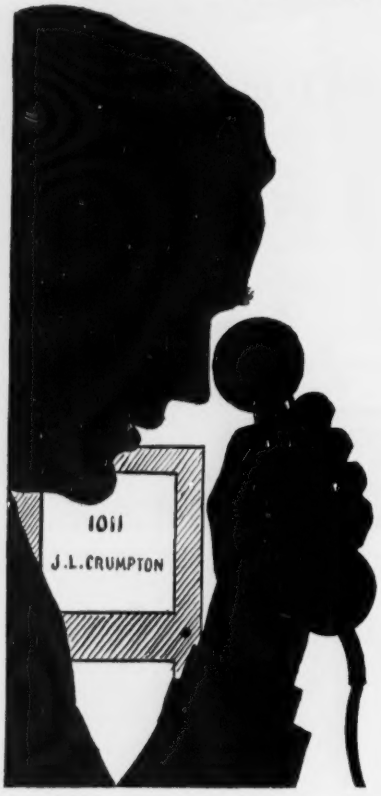

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[Contribution from the Research Laboratories of Parke, Davis and Co.]

THE ACTIVE PRINCIPLES OF THE POSTERIOR LOBE OF THE PITUITARY GLAND.[1] I. THE DEMONSTRATION OF THE PRESENCE OF TWO ACTIVE PRINCIPLES. II. THE SEPARATION OF THE TWO PRINCIPLES AND THEIR CONCENTRATION IN THE FORM OF POTENT SOLID PREPARATIONS

By Oliver Kamm, T. B. Aldrich, J. W. Grote, L. W. Rowe and E. P. Bugbee

Received December 31, 1927 Published February 4, 1928

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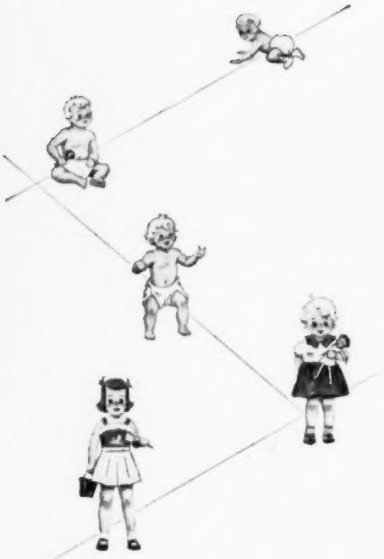

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